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HUMAN HEALTH RISK ASSESSMENT FOR HERBICIDE APPLICATIONS TO CONTROL NOXIOUS WEEDS AND POISONOUS PLANTS IN THE NORTHERN REGION
1988 EDITION

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SUMMARY OF THE RISK ASSESSMENT

Introduction

This summary discusses the risk to human health associated with herbicide application projects to control noxious weeds and poisonous plants on lands administered by the USDA Forest Service in the Northern Region. The Northern Region consists of approximately 24.8 million acres of National Forest System (NFS) land in northern Idaho, Montana, North Dakota, and western South Dakota. On a typical year, the Forest Service applies from 4,000 to 7,000 pounds of herbicide active ingredient on 5,000 to 10,000 acres of noxious weeds in the Northern Region. These treated areas constitute less than 0.05 percent of the total Forest Service administered land.

Public concerns over the possible human health and environmental impacts of pesticide use have prompted the Forest Service to examine the effects of pesticide use. This risk assessment represents part of the analytic process and is intended to be used in conjunction with the site-specific analysis and scoping process as required by the National Environmental Policy Act (NEPA).

Risk Assessment Methods

Human health risk assessment has been described as a three-part process comprised of an exposure analysis, a hazard analysis, and a risk analysis. The exposure analysis estimates the exposure and doses of the affected populations. The hazard analysis reviews the toxic characteristics of the chemicals of concern. The risk analysis compares human doses projected in the exposure analysis to the health-effect levels determined in the hazard analysis.

In this risk assessment, the exposure analysis is based on 5 project scenarios that are designed to encompass the range of projects sponsored by the Forest Service. Three open-range/forest projects (ground application) range in size from 1 treated acre through 40 treated acres up to large projects of 500 acres. A road right-of-way/riparian project of about 20 acres is described. In this scenario, the road is assumed to parallel a stream. Finally, an aerial application project of 120 acres is described, although aerial application for noxious weeds in this Region is unlikely.

Application rates of 1 to 2 pounds of herbicide active ingredient per treated acre are projected in this analysis. The analysis assumes that herbicide is sprayed over a continous area corresponding to the project sizes specified above. In reality, the application rates of many herbicides discussed here are lower than assumed in this analysis. The higher rates applied over the more confined areas assumed here serve to increase the estimated impacts of projects with lower herbicide application rates. For example, one-quarter pound of picloram applied per acre over 160 acres should have less impacts through drift and runoff to water than 40 pounds applied to 40 acres (assuming comparable buffers to sensitive resources).

A variety of mixing and application errors are also described that would increase estimated application rates by up to 20 percent.

In most cases, noxious weed control efforts will be far removed from human residences, thus drift impacts would be slight to nonexistent. However, no direct measurements have been made of nonworker populations in the vicinity of spray projects. Therefore, worst-case estimates are made of possible human exposures based on drift rates, dermal absorption, and other factors reported in the scientific literature. This analysis assumes that human residences are either 200 yards or 60 yards downwind of the projects and that residents are outside and exposed to drift while wearing little protective clothing. A variety of other conservative assumptions are made which increase estimated exposures from eating contaminated vegetables or cattle that have grazed on treated areas.

Worker-dose data are available for the herbicides 2,4-D, picloram, and dicamba. Worker doses for other herbicides are extrapolated from these studies based on similarities in dermal absorption rates of the herbicides. Sufficient data are available to estimate both average doses and high doses for workers. High doses would be expected 1 to 2 days out of 100. In addition, dose estimates are made for backpack sprayers who wear little protective clothing as well as those with recommended protective clothing.

When discussing the toxic characteristics of herbicides several toxicological terms must be defined. Important terms are discussed below as well as in a glossary at the end of the document. The acute toxicity of a chemical is often indicated by the one-time or short-term dose that is lethal to 50 percent of a group of treated animals. This value is abbreviated as the LD_{50} and is expressed as the amount of the compound (usually in grams, milligrams, or micrograms) administered per mass unit of the organism (usually in kilograms). The higher the LD_{50} the less toxic the compound.

The herbicide LD_{50} values for most species are in excess of 1,000 mg/kg which indicates relatively low acute toxicity. However, there are exceptions with some species/herbicide interactions. For example, the LD_{50} for dogs dosed with 2,4-D is 100 mg/kg.

The chronic toxicity of a chemical is typically determined by feeding low doses of the chemical daily for a large portion of the animal's lifetime. The animals are then sacrificed and examined for a wide variety of physiological and biochemical changes. In a properly conducted study, researchers can define a no-observed-effect level (NOEL) which is the highest dose level that does not affect organism health or well-being over the duration of the test.

In addition to studies of general systemic effects, more specialized studies probe the chemical's ability to disrupt animal reproductive functions or to cause fetotoxic or teratogenic effects (gross malformations) in offspring. Feeding studies can be conducted over several generations of animals. Pregnant animals can also be given high doses during critical times of pregnancy to test for teratogenic effects.

For many people, exposure to pesticides raises the specter of cancer or heritable mutations (alterations in genetic material that can be passed to succeeding generations as a "birth defect"). Carcinogenic potential can be tested in chronic oncogenicity (tumor) studies. Tests for mutagenic potential involve a variety of tests ranging from simple cellular organisms to mammalian studies. However, considerable scientific uncertainty exists as to what constitutes sufficient evidence of mutagenicity or carcinogenicity and the extent to which these herbicides constitute a threat to human health.

This analysis assumes as a worst case that a herbicide could cause cancer if any test evidence of carcinogenicity exists. The analysis also assumes that any dose of a carcinogen carries with it a probability of carcinogenic effect and that this probability increases with increasing dose. Thus, the analysis assumes that there is no absolutely safe dose of a carcinogenic compound.

A major issue in the Forest Service development of risk assessments to meet NEPA regulations has been the existence of data gaps on the health effects of herbicides. The U.S. EPA regulations for the registration of a pesticide for use on food require chronic and oncogenicity feeding studies on at least three animal species, in addition to a variety of tests for teratogenicity, reproductive effects, and mutagenicity. When all the data requirements for final registration have not been met, a pesticide can nonetheless receive a conditional registration provided there is no evidence of unacceptable risk to human health or the environment from its continued use. The critics of pesticide registration process often view this as a significant flaw in the system since it requires that EPA prove that the pesticide is unsafe rather than requiring that the manufacturer complete testing prior to use. In defense of the manufacturers, it should be noted that EPA has only recently issued final registration regulations. For most of the pesticides discussed here, a considerable body of test data exists, although all final test requirements may not be met.

With the possible exception of hexazinone, all of the pesticides discussed here are conditionally registered. Even though hexazinone completed the reregistration process several years ago and was granted a final registration, newer test requirements, such as a chronic-feeding study with a nonrodent species, have not been met for this compound.

For the remaining herbicides, the registration data gaps vary in their significance for a human health risk assessment. Glyphosate has met most of the registration data requirements, although the U.S. EPA has recently asked for additional data to clarify issues raised by a recent cancer study that showed a weak cancerous effect. Major chronic feeding studies with the herbicides 2,4-D and picloram are currently being reviewed by the EPA. Although at least two chronic-feeding studies with rats and mice are available for tebuthiuron, an additional nonrodent-feeding study is required.

The data gaps for amitrole and dicamba are more significant. Chronic feeding studies are not available for amitrole. However, the subchronic feeding studies are sufficient to indicate grounds for toxicological concerns. Many of the feeding studies available for dicamba were conducted in the 1960's. These earlier studies do not meet current standards for experimental protocol such as number of test animals. Subchronic feeding studies with dicamba are also being used by EPA in the interim.

Although the Forest Service spends approximately $800,000 per year through the National Agricultural Pesticide Impact Assessment Program on research projects to increase understanding of pesticide impacts, these funds are not sufficient to fill many of the health effects data gaps identified above. A chronic feeding study, for example, typically exceeds $1 million in cost. In addition, the herbicide manufacturers are under mandate by EPA to fill these data gaps. In the interim, worst case assumptions have been used where data is unavailable and unobtainable.

Sections 2.5 and 2.7 review in detail the toxicity data available on these herbicides. The NOELs used in this analysis range from 0.025 milligrams/kilogram/day for amitrole to 10 mg/kg/day for glyphosate and hexazinone. Amitrole, atrazine, 2,4-D, glyphosate, and picloram are assumed to be carcinogens.

Regardless of the status of health effects tests designed to meet EPA registration requirements, a major source of uncertainty is the extrapolation of the results of animal tests to humans. In order to compensate for the uncertainty of extrapolating from animals to humans, the EPA defines the acceptable daily intake (ADI) of a pesticide by dividing the NOEL for the species most sensitive to the pesticide by a safety factor. Safety factors of 100 are typically used when adequate chronic feeding studies are available. This risk assessment compares worker doses and combinations of general population doses to the NOEL and ADI levels to determine the significance of these doses. The comparison with ADI's will tend to overstate the possible impacts of Forest Service spraying since resulting exposures tend to be short term, whereas the ADI assumes a lifetime of such doses.

The carcinogenic potential of herbicides to humans is extrapolated from animal studies by use of a conservative model that tends to overestimate possible impacts. This estimate of the possible cancer impacts on humans is uncertain because of the differences between test animals and humans and the extrapolation of the results of high-dose animal studies to the low doses experienced by humans.

The final issue we will discuss in this section on herbicide toxicity is the bioaccumulation and biomagnification of pesticides in the environment. Much evidence exists of environmental damage by pesticides such as DDT, endrin, dieldrin, etc., which readily bioaccumulate in the environment and can damage organisms high on the food chain such as raptors (hawks, eagles, etc.) and possibly humans. Concern is expressed that the widespread use of these herbicides could have similar effects.

Bioaccumulation or biomagnification of the herbicides analyzed here is not possible. A chemical can bioaccumulate significantly only if it is persistent (that is, it does not degrade quickly) and it is insoluble in water (or conversely, high in lipid or fat solubility). Fortunately most of these herbicides are quite soluble in water and are excreted quickly by organisms. Although ester forms of 2,4-D are soluble in organic solvents indicative of lipid solubility, they degrade very rapidly in the environment and are excreted quickly by organisms. In summary, extensive testing of these herbicides indicates no significant bioaccumulation or biomagnification.

Comparison of ADI's and NOEL's with General Population Doses

Relatively low risk exists for the general public from herbicides widely used for noxious weed control. With but few exceptions discussed below, the worst-case doses to maximum-exposed members of the general population are all below ADI values.

Amitrol use provides the most potential for adverse human impacts. Amitrole could be a potent antithyroid agent in humans as evidenced by the low subchronic feeding levels (2 ppm in the diet or 0.1 mg/kg by body weight in rats) that result in significant effects on thyroid function. Doses to maximum-exposed residents could be 12 to 20 times lower than the NOEL for amitrole based on thyroid effects. Doses to visitors to a spray site who eat wild foods sprayed with amitrole could exceed the NOEL.

As discussed in Section 2.5 of the Risk Assessment, on a body-weight basis, humans can be as much as 6 to 12 times more sensitive to effects of chemicals than smaller test animals. Since the antithyroid effects of amitrole can be exhibited in a relatively short time (less than 90 days' dose), the general population would be at risk from worst-case doses.

Several mitigating measures can greatly reduce the possibility of adverse impacts on the general public from amitrole application. Ensuring that cattle do not graze in treated areas will reduce the possibility of secondary doses to humans consuming beef. Posting amitrole-sprayed areas to warn the public against consuming wild foods on-site will reduce the possibility of these oral doses. Ensuring that spray sites are at least one-half mile from all residences, food crops, and gardens will ensure that drift-related doses are less than 5 percent of worst-case doses calculated in the risk assessment. Careful site-specific analysis would be needed for any proposal to use this herbicide. At this time, use of this compound in the Northern Region is unlikely.

The NOEL/dose and ADI/dose comparisons for the remaining herbicides show that the exposure with the highest risk to the general public would involve comsumption of wild food from a spray site. The dose comparisons show that a visitor to National Forest System lands can receive a dose that exceeds the ADI's if he or she collects and consumes a large quantity of sprayed, unwashed vegetation. For numerous reasons, there is a very low probability of this event. Very little land would actually be sprayed for noxious weeds (less than 0.04 percent of National Forest System land per year in the Northern Region). The targeted vegetation is not edible and berry bushes and other prime food-gathering areas generally do not occupy the same habitats that are infested with noxious weeds or poisonous plants. Finally, the appearance, odor, and taste of the sprayed vegetation would significantly reduce palatability of wild foods. Nonetheless, the calculated worst-case doses indicate that even if these improbable events were realized, the dose would be from 20 to 1,200 times less than the herbicide NOEL's based on long-term feeding studies.

The maximum estimated dose to an adolescent who spends the day in the vicinity of a right-of-way spray project could exceed slightly the ADI for the herbicide 2,4-D. This dose would be about 95 times lower than the NOEL based on long-term dosing studies. Since the adolescent dose would occur at most only once at the levels predicted, health effects would be unlikely.

Doses to members of the general population from atrazine-treated, open-range projects could exceed the ADI for atrazine if the person consumes a large amount of beef that grazed exclusively on atrazine-treated range. Label restrictions prohibit grazing cattle on atrazine-treated range for 3 to 7 months after treatment depending on application timing. The Forest Service would enforce these label restrictions to prevent this exposure. In addition, the small amount of atrazine applied in a year on scattered sites ensures that doses on the order estimated in the analysis are virtually impossible.

Under routine conditions, the aerial application of herbicides would not significantly increase public exposure over that received during ground application. However, misapplications and accidents during aerial application could increase public exposure. For example, if a plane directly sprayed a small stream with 2,4-D and an adult immediately drank a liter of water from this stream, his dose would exceed the ADI for 2,4-D. Generally, the doses from exposure to all other aerially applied herbicides (picloram, dicamba, and tebuthiuron) are below the ADI's except in the case of a direct spray or spill of herbicides over bystanders.

Although most doses to the general public are well below NOEL and ADI levels based on animal tests, it is possible that a small percentage of the human population may be very sensitive to chemical exposures. For example, the medical literature has reported several cases of peripheral neuropathy resulting from exposure to 2,4-D. Peripheral neuropathy is the disruption of the nervous system characterized by some or all of the following symptoms: numbness in hands and feet, loss of balance, aching in extremities, fatigue, and nausea. Recovery in some cases is very prolonged and may not be complete even after several years.

Although most of the reported cases of pheripheral neuropathy occurred after massive doses, effects in some people have been noted at much lower apparent doses. The conclusions of Berkley and Magee (1963) seem appropriate.

> "Despite the extensive use of 2,4-D preparations, resultant peripheral neuropathy is very rare, and an affected individual probably has some predisposition to neuropathy or susceptibility to the toxin. Nevertheless, as it cannot be determined who is predisposed or susceptible, and as no antidote to 2,4-D intoxication is known, prevention is simpler than treatment."

It is also possible that idiosyncratic toxic responses as yet undetected by the medical community could result from the exposure to other herbicides or combinations of herbicides. Once again, prevention of exposure for both workers and the general public is the most prudent course.

Comparison of ADI's and NOEL's with Worker Doses

In general, of the various populations exposed to pesticides, the workers applying herbicides incur the highest risk of health impacts. The workers with the highest potential exposure are backpack sprayers. This section will summarize the trends in worker doses for each herbicide.

Amitrole

Amitrole application could pose a risk of thyroid effects based on a comparison of worker doses with dose levels that disrupt thyroid functions in animals. Although an ADI for amitrole has not been set by EPA because it is not approved for use on food or feed, the proximity of most worker dose estimates to the amitrole NOEL level is cause for concern. Workers applying amitrole with careless techniques and little protective clothing for relatively short periods of time (perhaps as little as 7 to 30 days) stand a significant risk of disrupting thyroid function.

Measures that could reduce human health impacts to workers include use of rubber gloves and rubber boots, careful application techniques, and personal hygiene to avoid dermal and oral exposure. Limitations on worker exposure to amitrole to less than 5 days per year would also reduce the probability of cumulative impacts on thyroid function although data are not available to indicate whether the risk could be eliminated.

Even with allowances for protective clothing, the average worker dose is below the NOEL by only a factor of five. As noted above, humans can be 6 to 12 times more sensitive on a body weight basis than test animals. For example, it is possible that a chemical that affects animals at a dose of 10 mg/kg/day, could elicit the same response in humans at 1 to 2 mg/kg/day. Thus, worker doses under many conditions examined in this assessment could effect health.

Worker dose under all protective scenarios are at least 100 times less than dose levels that cause fetotoxic effects (toxicity to fetuses).

Atrazine

Assuming good protective techniques and careful application habits, worker doses from atrazine applications can be at or below the ADI for atrazine. Conversely, careless techniques could result in doses that are as little as 25 times lower than the NOEL. Worker doses, under all protection scenarios, are at least 100 times less than dose levels causing fetotoxic effects in humans.

2,4-D

All worker dose estimates for backpack applicators, truck applicators, pilots, and mixer/loaders are above the ADI for 2,4-D. Backpack-applicator doses range from as little as one-third the NOEL levels to 24 times less than the NOEL. At dose levels above the NOEL, changes in kidney function in test animals were observed with as little as 90 days' dosing. Tests on the reversibility of the changes in kidney function have not been conducted. Backpack applicators are at some risk of effects on kidney function. Application techniques and the use of rubber gloves, rubber boots, and long-sleeved shirts can reduce this impact.

The dose estimates for truck applicators, pilots, and mixer/loaders are lower than backpack applicator dose estimates, thus the risk of kidney effects is lessened although not completely eliminated. For all worker functions, there exists the possibility of neuropathological effects as described above in the general population section.

Worker doses (backpack applicators, truck applicators, pilots, and mixer/loaders) range from 80 times less under the fetotoxic NOEL under low protection scenarios to 675 times less under recommended protection scenarios.

Dicamba

Of the herbicides analyzed here, dicamba poses the greatest risk of fetotoxic or reproductive effects for pregnant applicators. The NOEL of 2.5 mg/kg/day was based on observed disruption of reproductive functions in female rats. Dose estimates for backpack workers, truck applicators, pilots, and mixer/loaders range from 16 to 1,250 times less than this NOEL.

A NOEL of 25 mg/kg/day has been established for general systemic toxic effects (kidney and liver function) based on a 90-day feeding study. Thus, the risk of adverse health effects would be much lower for male applicators. NOEL/dose comparisons would range from 160 to 12,500.

Glyphosate

Except for the high dose estimate for backpack applicator under the low protection scenario, all worker dose estimates are below the ADI for glyphosate.

Hexazinone

NOEL/dose comparison factors for hexazinone application under recommended protection scenarios are all at least 100. Under low protection scenarios, the high doses could be as little as 32 times less than the NOEL.

The fetotoxicity NOEL for hexazinone is 50 mg/kg. This NOEL is at about 200 times higher than the highest worker dose estimate for any project type and protection scenario.

Picloram

Picloram is similar to glyphosate in that all worker dose estimates are below the ADI except the highest dose estimate under the low protection backpack applicator scenario. The NOEL for general systemic effects is about 226 times higher than this high-dose estimate.

Tebuthiuron

Because tebuthiuron is applied in pellet form, the worker exposure to drift and herbicide-treated foliage is minimized. All worker dose estimates are below the ADI for tebuthiuron.

Risk of Cancer and Mutation

Based on extensive testing of a large number of both synthetic (man-made) and naturally occurring chemicals, it is apparent that carcinogens vary widely in their potency. Nor is there a reliable or universal difference in the carcinogenic potential of either "natural" or "synthetic" chemicals. For example, over a lifetime a daily dose of about 2 micrograms of aflatoxin B1 is sufficient to give 50 percent of test rats cancer. Aflatoxin B1 is a naturally occurring compound in milk, peanut butter, cornmeal, and other foods. By contrast, it requires daily doses of approximately 1 million micrograms of saccharin to induce cancer in 50 percent of test animals.

Although cancer-causing chemicals can vary tremendously in their cancer potencies, it is not known if there are "cancer NOEL's"--that is, dose levels that are absolutely free of cancer risk. If million-microgram daily doses give 50 percent of test animals cancer, and half-million microgram daily doses give 10 percent of animals cancer, what can we say with certainty about 10 microgram doses? Not much, it turns out. It is possible that if a million animals were dosed daily with 10 micrograms of Chemical X, one or two animals might develop cancer as a result of these low doses. Since we have no way of testing a million animals, we cannot resolve the issue given the current state of science. In the face of this uncertainty, this risk assessment assumes that any dose of a carcinogen above zero carries with it a possibility of causing cancer. For low doses of weak carcinogens, this possibility is often expressed as the chance of developing cancer. For example, if a particular dose is said to bring a five-in-a-million chance of cancer, it is equivalent to saying that if a million people received the dose we would expect five to develop cancer.

Of the herbicides under consideration, testing of tebuthiuron, hexazinone, and dicamba indicate no carcinogenic potential. Of the remainder, amitrole has been classified by EPA as a probable human carcinogen. A recently completed atrazine cancer study indicated a significant carcinogenic effect in test animals. This study is currently being reviewed by EPA.

For the herbicides glyphosate, picloram, and 2,4-D, scientific uncertainty exists regarding their carcinogenicity. Animal feeding studies conducted to date indicate that carcinogenic effects, if any, are weak. The cancer potencies of these herbicides are on the order of saccharin. For example, at the highest dose levels in which 3 percent of the test animal diet was glyphosate, only three of 50 male rats developed cancer and no females developed cancer.

In addition to animal feeding studies, numerous human epidemiology studies have been conducted on phenoxy herbicides including 2,4-D. Epidemiology is the study of the relative rates of disease in populations that have been exposed at different levels to potential disease-causing agents. A recent National Cancer Institute (NCI) study of farm worker exposure to various herbicides reports that farmers who were exposed to phenoxyacetic acid herbicides (including 2,4-D) were about twice as likely as nonfarmers to develop Non-Hodgkins Lymphoma (NHL), a rare form of cancer. Farmers who used personal protective measures were about 1.4 times as likely as nonfarmers to develop NHL. Farmers who did not protect themselves were about 2.1 times as likely as nonfarmers to develop NHL. Farmers who were exposed to phenoxyacetic acids over 20 days per year increased their odds of NHL sixfold. Farmers who were exposed over 20 days per year and did their own mixing and loading showed an eightfold increase. The NCI study found no increase in NHL rates in farm family members who were not actively involved in herbicide applications.

Some findings of this study were inconsistent. For example, although farmers with increased exposure days per year had increased rates of NHL, there was no relation between increased acres sprayed per year and increased NHL rates. Likewise, an increase in the number of years of spraying was not associated with an increase in NHL rates. These inconsistencies may be reconciled by additional ongoing studies currently being conducted by the NCI.

At the present time EPA has not initiated a special review of 2,4-D based on the NCI findings. EPA has noted that several other epidemiology studies have not replicated the positive findings of the NCI study. On the other hand, it should be noted that several European epidemiology studies of workers exposed to a variety of chemicals including 2,4-D have found associations with cancers in addition to NHL. While ongoing studies are not completed, the epidemiologic findings to date and the results of animal testing studies certainly argue, at a minimum, for care in the use of 2,4-D to reduce exposure.

Based on the extrapolation of the results of animal cancer studies to humans, it is again apparent that workers are at highest risk. A lifetime of 2,4-D exposure (assuming 30 days of application per year for 30 years) could increase the worker's chances of getting cancer by about five chances in 100,000. Since the average American has about one chance in four of cancer regardless of herbicide exposure, this cancer increase is still rather small. Animal studies indicate that amitrole and atrazine are 10 to 100 times more potent carcinogens than 2,4-D; thus, doses comparable to 2,4-D would proportionately increase cancer risk. However, use of these compounds would be extremely limited and comparable exposure is unlikely. Picloram and glyphosate are 10 to 100 times less potent carcinogens than 2,4-D based on animal feeding studies. Thus, comparable doses would result in proportionately less cancer risk.

Cancer risks to members of the general public are 100 to 1,000 times less than the risk to workers when considering exposure to the same herbicide. Risks on this order could not be detected by epidemiology studies as conducted by the National Cancer Institute. As noted above, the NCI study did not find increased cancer in farm family members that were not actively involved in pesticide application.

Finally, as a point of comparison, Table S-1 presents the risk of death or cancer from a variety of other activities Americans engage in.

Cumulative and Synergistic Effects and Inert Ingredients

The possible interaction of pesticide active ingredients with other chemicals in the environment has been raised as an issue. Of particular concern is the possibility of synergism, a special type of interaction where the combined effect of a specific herbicide with one or more chemicals in the environment (such as pollutants) would be greater than the sum of the individual effects of the herbicide and chemical(s) (in other words, 2 + 2 are greater than 4).

A classic study of the synergistic effects of pollutants examined the interactive effects of asbestos exposure and smoking. This study found that inhalation of cigarette smoke and asbestos resulted in an eightfold increase in lung cancer over nonsmokers exposed to only asbestos. Studies such as these, however, have limitations because high doses are required to discover effects and the relevance to low-level exposures is uncertain.

Some mixtures of picloram and 2,4-D are reported to cause dermal sensitization in a small percentage of the human population, and precautionary statements to this effect are contained on the label for Tordon 101 (trademark for a mixture of the triisopropanolamine salts of picloram and 2,4-D). Since these compounds are not reported to cause dermal sensitization reactions singly, there may be an interactive effect from this mixture. Tank mixes (i.e., mixed in the field) of the potassium salt of picloram and the amine salts of 2,4-D have not been reported to cause dermal sensitization in the field, but such mixtures have not been specifically studied in the laboratory.

In the process of formulating pesticides for commercial use, a variety of surfactants, emulsifiers, diluents, and other so-called inert ingredients may be added. The toxicological properties of these additives have come under increased scrutiny. EPA has issued two lists of inerts requiring further regulation or testing. The first list of about 55 chemicals groups the "Inerts of Toxicological Concern," and a second list of 60 chemicals are "Potentially Toxic Inerts/High Priority for Testing."

Some formulations of 2,4-D contain petroleum distillates which is a class of chemicals found on List 2. Analysis of the health risk of these petroleum products indicates that these inerts pose less risk than the active ingredients. Tests of the acute toxicity of pesticide formulations support this contention. The LD_{50} values for the pesticide formulations are typically higher than those of the active ingredient, indicating that the formulations are less toxic. Unfortunately, chronic tests of pesticide formulations are not available and interactive effects on cancer rates or other health effects cannot be ruled out absolutely.

In summary, then, what can be said concerning the issue of synergistic and cumulative effects of herbicides used in Forest Service herbicide plant control programs?

First, the additive impact of Forest Service spraying relative to the effects of the private application of herbicides will be very small. For example, a worker or farmer who sprays herbicides on non-Forest Service projects and is also a resident in the vicinity of Forest Service projects might expect, under worst-case conditions, an increase in herbicide dose of about 1 percent over his worker dose. Typically, the increase would not be measurable.

Second, the total doses to members of the general public from all sources of herbicides are unlikely to be higher than those estimated in these analyses. The dose to maximum-exposed residents assumed that the greatest portion of their diet came from spray-impacted foodstuffs. Any substitution of food from other sources (i.e., food markets) would lessen the dose. The herbicides involved in these analyses have not been found widely in market foodstuffs. For example, a market-basket analysis by the Natural Resources Defense Council (NRDC) of a variety of fruits and vegetables found no 2,4-D in any food sample.

Although the NRDC found other pesticides in some foodstuffs, the interactive effects would be suspected to be small for maximum-exposed residents. Dr. Bruce Ames, an eminent toxicologist from the University of California at Berkeley, has pointed out that there are many naturally occurring chemicals in the food that people eat that are teratogenic, mutagenic, and carcinogenic, and which are consumed at doses 10,000 times higher than man-made herbicides (see discussion in Ames 1983). Therefore, the low, short-lived doses to maximum-exposed residents that result from spraying of these herbicides are very small compared to many other chemicals in the environment. For these small comparative doses, a synergistic effect is not realistically expected.

As discussed throughout these analyses, the highest doses are expected of some types of workers, particularly those involved in the hand application of herbicides. In some cases, workers may apply mixtures of herbicides, particularly 2,4-D/picloram. If one assumed synergistic reactions on the order of those observed in the case of asbestos exposure and smoking, then eight to tenfold increases in herbicide toxicity might be expected. Cancer risk from these herbicides could then be increased accordingly.

Major Accident Scenarios

An examination of accident records for the past 10 years reveals no major accidents involving herbicide application projects. The possibility of accidents in the future cannot be completely discounted, however.

Several accident types including spills of concentrated herbicide formulations onto people or into drinking water reservoirs are reviewed. Spills of concentrate onto people could cause acute effects including nausea, trembling, headache, etc., depending on the degree of exposure, time to cleanup, and individual factors.

Spills into drinking water reservoirs would present less risk to individuals primarily because relatively little herbicide is carried at any one time and any spilled amounts would be quickly diluted. Two reservoir sizes were modeled. Concentrations in the larger reservoir would never be high enough to exceed ADI levels. Within a day in the smaller reservoir, levels would be below levels exceeding the ADI for water consumers.

Table S-1.--Lifetime risk of death or cancer resulting from everyday activities.

Activity	Time to accumulate a one-in-a-million risk of death	Average annual risk per capita
	Living in the United States	
Motor vehicle accident	1.5 days	2×10^{-4}
Falls	6 days	6×10^{-5}
Drowning	10 days	4×10^{-5}
Fires	13 days	3×10^{-5}
Firearms	36 days	1×10^{-5}
Electrocution	2 months	5×10^{-6}
Tornados	20 months	6×10^{-7}
Floods	20 months	6×10^{-7}
Lightning	2 years	5×10^{-7}
Animal bite or sting	4 years	2×10^{-7}
	Occupational Risks	
General		
manufacturing	4.5 days	8×10^{-5}
trade	7 days	5×10^{-3}
service & government	3.5 days	1×10^{-4}
transport & public utilities	1 day	4×10^{-4}
agriculture	15 hours	6×10^{-4}
construction	14 hours	6×10^{-4}
mining and quarrying	9 hours	1×10^{-3}
Specific		
coal mining (accidents)	14 hours	6×10^{-4}
police duty	1.5 days	2×10^{-4}
railroad employment	1.5 days	2×10^{-4}
fire fighting	11 hours	8×10^{-4}

Other One-In-A-Million Risks

Source of risk	Type and amount of exposure: examples
Cosmic rays	One transcontinental round trip by air; living 1.5 months in Colorado compared to New York; camping at 15,000 feet over 6 days compared to sea level.
Other	20 days of sea level natural background radiation; 2.5 months in masonry rather than wood building; 1/7 of a chest x-ray using modern equipment.
Eating & drinking	40 diet sodas (saccharin) 6 pounds of peanut butter (aflatoxin) 180 pints of milk (aflatoxin) 200 gallons of drinking water from Miami or New Orleans 90 pounds of broiled steak (cancer risk only)
Smoking	2 cigarettes

[1] From Crouch and Wilson (1982)

1.0 INTRODUCTION

This document analyzes the risk to human health associated with herbicide application projects to control noxious weeds and poisonous plants within the Northern Region of the USDA Forest Service. The cumulative impacts of treatment programs are also discussed. This document is not intended to replace the site-specific analysis and scoping process required by the National Environmental Policy Act (NEPA). However, the analyses in this document provide a point of comparison for specific projects, and information in this document can be incorporated into the NEPA process.

Human health risk assessment has been described as a three-part process comprised of an exposure analysis, a hazard analysis, and a risk analysis (National Research Council 1983). Exposure analysis involves estimating the exposure and dose of affected populations. Hazard analysis involves reviewing the toxic characteristics of the chemicals of concern and developing dose-response relationships for these chemicals. Risk analysis involves comparing dose levels determined in the exposure analysis to the effect levels determined in the hazard analysis.

The terminology described above can be confusing. For example, the term "risk analysis" is often used to describe the overall analytic process as well as one element in the process. The terms "risk" and "hazard" are frequently used interchanageably in common parlance.

The analyses provided in this document have been further divided into the following sections:

1. Identification of the characteristics of typical herbicide application projects including sizes of spray areas, locations of spray areas, herbicide application rates, and application methods.

2. Identification of the problems, misapplications, and accidents that are possible with herbicide spraying projects and a determination of the probabilities of these events.

3. Identification of the human population potentially affected by herbicide application programs (population at risk).

4. Estimation of the exposure and dosage of the affected populations based on the project characteristics outlined in steps 1 through 3.

5. Review of the health effects data including the general toxic effects of the compounds of interest. Identification of dose-response relationships, no-effect dose levels, and the potential of certain herbicides to cause cancer and mutagenic effects.

6. Comparison of dose levels (from Step 4) with toxic-effect levels for which safety thresholds can be established (from Step 5).

7. Discussion and determination of the probability of irreversible effects (cancer and heritable mutations) for which absolute safety thresholds cannot be assumed for the population at risk.

Steps 1 through 4 correspond to an exposure analysis. Step 5 could be described as the hazard analysis, and steps 6 and 7 the risk analysis.

Each of these steps will be discussed briefly in the introduction and in greater detail in the main body of the report. Sections 1.1 through 1.7 and Sections 2.1 through 2.7 correspond topically to the seven discussion areas.

1.1 Overview of Northern Region Weed Control Program

The Northern Region consists of approximately 24.8 million acres of National Forest System (NFS) land in northern Idaho, Montana, North Dakota, and northwestern South Dakota. In 1987, the USDA Forest Service applied about 7,400 pounds of herbicide active ingredient to about 7,300 acres of noxious weeds and poisonous plants in this Region. This figure may increase in the next several years as additional forests initiate treatment programs. However, budget constraints or the development of biological control techniques could limit this increase or even reduce the pesticide treatment program.

Treatment projects range in size from less than 1 acre to several hundred acres. Generally treatment projects are relatively small because the Forest Service strategy has been to reduce the spread of noxious weeds rather than eradicate large, firmly established infestations. Noxious weed treatment projects in the past have primarily involved ground application of the herbicides 2,4-D, dicamba, and picloram.

The possible adverse impact of individual projects is a primary concern in these analyses. Therefore, various model projects that represent segments of the entire program acreage for the Region are analyzed. These models are intended to encompass the range of typical Forest Service application practices in the Northern Region. These models are based on typical application rates, typical spray acreages and application methods, and a variety of locational variables. These models also form the basis on which the effects of deviations from standard procedures can be assessed as outlined in the next section.

1.2 Errors and Misapplications Associated with Herbicide Spray Projects

There is associated with any human activity the probability of errors which can affect various human populations. Mixing errors and excess application can increase human exposure and are assumed in the model projects, as outlined in Sections 1.1 and 2.1. In addition, the drift of herbicide spray is considered intrinsic to the application process even with ground application methods.

The probability of errors and accidents is difficult to determine. Calculations in these analyses are based on incidence reports where available and, where necessary, application of statistical probability functions to determine the upper limits of the accident rate. Mixing errors resulting in excess concentration of herbicides in field mixtures are assumed to occur to the point that impacts on herbicide utilization would be noticed (see Section 2.2).

The potential impacts of catastrophic accidents (e.g., large truck spills) are analyzed separately in Section 3.

1.3 Affected Human Populations

Two populations are considered in this analysis: workers and the general public. The first includes the group of operators, supervisors, and other personnel directly involved in the application of herbicides to control target plants. The second population includes the members of the public who could contact the herbicide in spray drift, spills, and on sprayed vegetation or through the consumption of contaminated water, vegetation, fish, or meat from herbivores. Possible impacts on fish, wildlife, livestock, and nontarget plant species are considered in these human health risk analyses insofar as they affect human consumers.

1.4 Affected Human Population Exposure and Dosage

The determination of the exposure level and dosage of the affected population is based on several sources of information. Several studies have measured herbicide doses received by pesticide applicators and these findings are applied in these analyses. In some cases, estimates of the doses received by the general population have been extrapolated from worker data in order to analyze possible worst-case impacts. In other cases, doses have been calculated based on maximum drift rates, dermal exposure and absorption rates, and food intake rates.

1.5 General Toxic Effects of Herbicide Exposure

The general toxic effects of each herbicide are reviewed in this document. The LD_{50} values (lethal dose to 50 percent of a given test population) for each chemical have been reviewed to determine its relative acute toxicity. The "no-observable effect levels" (NOEL), for chronic exposure to a chemical were also reviewed. Both LD_{50} and NOEL values are provided in these analyses for the animal species found to be most sensitive to each herbicide. The effects of chemical doses above the NOEL are noted.

This document provides acceptable daily intake (ADI) values for the herbicides of interest as determined by U. S. Environmental Protection Agency (U.S. EPA) review of the toxicity data for these compounds in the herbicide use registration process. ADI's are based on NOEL values using safety factors of 100 or greater.

1.6 Comparisons of Dose and Effect Levels

The dose levels to maximum-exposed members of the affected population are compared to NOEL and ADI values for each of the herbicides of interest. This comparison indicates the likelihood of adverse human health impacts from the maximum calculated doses. Possible effects of doses that exceed the ADI and approach the NOEL are discussed in this section.

1.7 Carcinogenic and Mutagenic Effects

A separate discussion of the carcinogenic and mutagenic potential of herbicide doses is provided in these analyses. As noted in Section 2.7, amitrole is a demonstrated animal carcinogen and has been designated by EPA as a probable human carcinogen. Questions have been raised concerning the possible carcinogenicity of 2,4-D, picloram, atrazine, and glyphosate. This analysis assumes that a herbicide is a carcinogen if any animal or human study data indicate carcinogenic activity, no matter how weak. The probability of a human carcinogenic response from the maximum doses is calculated in Section 2.7. These calculations are based on the animal test data and use a conservative predictive model that tends to overestimate the possible incidence of cancer.

1.8 Worst-Case Analysis Requirements and Notes on Data Sources

A variety of data sources are used in these analyses. An important source is Forest Service Agriculture Handbook No. 633 and its supplements (USDA, Forest Service 1984 and 1986a). Handbook No. 633 summarizes the extensive data on the human health effects and the environmental effects of several herbicides, including the herbicides of interest here. Handbook No. 633 can be inspected by contacting the pesticide coordinator at the Forest Supervisor Office for any National Forest in the Northern Region or at the Regional Office in Missoula, Montana.

Although Handbook No. 633 (USDA, Forest Service 1984 and 1986a) summarizes human health and environmental effects data, it was beyond the scope of this Handbook to critically evaluate the field and laboratory data upon which conclusions were reached in the studies reported. Therefore, all herbicide health effects data used in this analysis were cross checked against health effects data used in the U.S. EPA herbicide registration process. Because U.S. EPA extensively reviews the data used in support of the registration process, the health effects data were also discussed with the U.S. EPA toxicologists who review toxicology data for each of the herbicides. In addition, the progress and implication of ongoing studies were reviewed with these toxicologists.

Much of the general environmental fate data on these herbicides was collected by investigators at universities, government agencies, contract laboratories, and industry laboratories. This data base is extensive for the herbicides of interest. Values were selected from the data pool that would increase projected human health impacts. Selection of data that increase projected human impacts thus ensures that any inaccuracies contained in the data used in this analysis err on the side of overestimating possible adverse human health effects.

Although a considerable amount of data is available on the herbicides analyzed here, it is important to note that, with the exception of hexazinone, these herbicides have received only conditional registration. A pesticide is registered conditionally when additional data is required to reregister the pesticide under Sections 3(g) and 3(c)(2)(B) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Thus, in terms of EPA's registration process, data gaps exist for these pesticides. In addition, a variety of test data not currently required by EPA would also be desirable in conducting risk assessments.

The Council on Environmental Quality (CEQ) regulations for Environmental Impact Statements contain several requirements when information essential for a reasoned choice among alternatives is unavailable or unattainable. Because the analysis provided here is a basis for additional site-specific analyses in compliance with NEPA, CEQ regulations and applicable case law regarding missing information were followed.

This risk assessment identifies a number of areas of uncertainties or data gaps including the following:

1. Field studies on worker doses for all of the herbicides except 2,4-D, dicamba, and picloram.
2. Information on exposure of the public to the eight herbicides.
3. Field data on residue levels in plants and animals most likely to be found in and around treatment areas for some of the herbicides.
4. The carcinogenic potential of amitrole, atrazine, 2,4-D, picloram, and glyphosate, particularly at doses lower than tested in the laboratory.
5. Toxicological information on inert ingredients contained in pesticide formulations.
6. Toxicity information on the synergistic effects from exposure to more than one herbicide.
7. The extrapolation of the results of animal toxicity tests to exposed humans.

These areas of uncertainty are important in deciding what is the best alternative for action; however, the cost of obtaining this information is an important consideration. From discussions with the Environmental Protection Agency, the Department of Agriculture, the Department of the Interior, and chemical manufacturers, it is estimated that the costs per chemical of conducting some of the standard laboratory toxicity tests would be $1.5 to $2.0 millon for a chronic-toxicity study with rats and dogs; $1.5 to $2.5 million for an oncogenicity test with rats and mice; and $50,000 to $100,000 for each mutagenicity and chromosomal study.

The estimated costs to fill the specific data gaps listed above are:

1. Worker exposure and dose studies would cost approximately $200,000 per chemical.

2. Studies measuring all routes of exposure of the public would be more expensive than the worker exposure studies.

3. The cost of measuring residues in plants and animals would be between \$50,000 and \$100,000 per chemical per plant or animal.

4. Additional oncogenicity (cancer) studies for these herbicides would cost at least \$20 million.

5. Chronic-toxicity studies for amitrole (two species) would cost \$3 to \$4 million.

6. Synergistic studies would be extremely expensive because of the great number of tests that would be necessary; there are 28 combinations of the herbicides if studied two at a time.

Currently the Forest Service funds research to fill data gaps in our understanding of pesticides through the National Agricultural Pesticide Impact Assessment Program (NAPIAP). A variety of research efforts are being funded including worker-exposure studies, residue studies in various segments of the environment, and studies of the impacts of pesticide use on wildlife. The NAPIAP budget for the past several years has been about \$800,000 per year.

The research costs to fill many of the data gaps outlined above greatly exceed the research funds available to the Forest Service. An independent attempt by Forest Service to fill data gaps such as the cancer studies outlined above would duplicate the efforts of other government agencies such as the EPA. Many of the missing toxicological studies have already been requested by EPA, and the results of these studies will be considered when they become available.

A worst-case analysis was conducted for those areas where information is currently unavailable or when there is uncertainty. The probability of a worst case occurring was also estimated. For example, the analysis estimated the upper limit of worker and public exposure from routine application procedures and from accidents. The model application projects combine parameters such as treatment unit size, duration of exposure, application rate, application equipment, and meteorological conditions that give the highest reasonable exposure value. Accidents include direct spills of concentrate on workers' skin, the direct spraying of an individual, and public exposure through drinking water contaminated by a spill.

The analysis of the mutagenic potential of chemicals assumed that the herbicide was mutagenic if positive tests occurred. The probability of mutagenic activity was based on available cancer data. Herbicides that had either positive cancer studies or for which there is scientific uncertainty were assumed to cause cancer. The cancer potency value for a chemical was computed by using the highest rates of tumor formation found in the available animal studies. A conservative model for predicting cancer rates in humans was also used. Synergistic effects were assumed to occur, although the probability of these effects was considered low. The data gaps currently existing for these pesticides and the methods for compensating for these data gaps are discussed in great detail throughout the following analysis. In addition, the large body of research available on these pesticides is reviewed as appropriate for this analysis.

1.9 Metric Usage and Scientific Notation

This document attempts to analyze risk as completely as possible with all assumptions clarified and intermediate steps and calculations explicitly detailed. As such, this document must cover issues and use analytical methods and terminology that are unfamiliar to the general public. When a concept is first developed, the document explains the terminology and methodology in terms that the average person can understand. In addition, a glossary that defines scientific terms used in this analysis is appended to this document.

Examples of all calculations are provided to allow the interested reader to track the development of dose factors and conclusions concerning possible human health impact. The metric system is used throughout in these calculations for several reasons. First, most of the scientific literature cited in this analysis uses the metric system. Second, a primary goal of this analysis is the calculation of the human dosage which is universally expressed in metric terms, typically as the milligrams or micrograms of compound absorbed by the person per kilogram of body weight. Thus, the calculations throughout this analysis are provided with greater ease in the metric system.

To assist the reader, English equivalents to metric units are provided in the text. Table 1-1 also provides a conversion for metric and English units.

Table 1-1. Conversions for English and Metric units.

1 cubic foot/second (CFS) = 28.3 liters/second
1 gallon (US) = 3.785 liters
1 gallon = 128 fluid ounces
1 gallon/acre = 9.354 liters/hectare
1 acre = .4 hectare
1 hectare = 2.471 acres
1 hectare = 10,000 square meters
1 milligram = 1,000 micrograms
1 gram = 1,000 milligrams
1 kilogram = 1,000 grams
1 kilogram = 1,000,000 milligrams
1 kilogram = 2.205 pounds
1 kilogram/hectare = 0.9 pounds/acre
1 kilometer = 0.62 miles
1 meter = 39.37 inches or 3.3 feet
1 liter/hectare = 0.107 gallons/acre
1 mile = 1.61 kilometers
1 fluid ounce = 29.573 milliliters
1 ounce = 28.35 grams
1 pound = 453.59 grams
1 pound = 0.453 kilogram
1 pound/gallon = 0.12 kilograms/liter
1 pound/acre = 1.12 kilograms/hectare
1 milligram/kilogram is equivalent to 1 part/million

Table 1-2 explains the use of scientific notation which expresses very large or very small numbers in powers of 10.

Table 1-2. Scientific notation.

$0.0000023 = 2.3 \times 10^{-6}$
$0.000023 = 2.3 \times 10^{-5}$
$0.00023 = 2.3 \times 10^{-4}$
$0.0023 = 2.3 \times 10^{-3}$
$0.023 = 2.3 \times 10^{-2}$
$0.23 = 2.3 \times 10^{-1}$
$2.3 = 2.3 \times 10^{0}$
$23.0 = 2.3 \times 10^{1}$
$230.0 = 2.3 \times 10^{2}$
$2{,}300.0 = 2.3 \times 10^{3}$
$23{,}000.0 = 2.3 \times 10^{4}$

2.0 ANALYSIS OF THE EFFECTS OF HERBICIDE SPRAYING

2.1 DESCRIPTION OF THE FOREST SERVICE SPRAY PROGRAM AND THE MODEL PROJECTS

Five model projects provide the basis for determining the human health impacts of the Forest Service program to control noxious weeds in the Northern Region. These models are based on the scope and design of the Forest Service control program as discussed below.

The total amount of herbicide sprayed annually by the Forest Service in the Northern Region will vary depending on the extent of noxious weed infestation, prognosis for other control techniques such as biological control, funding levels, and other factors. Regionwide spraying for weed control will typically involve 10,000 pounds or less herbicide active ingredient (a.i.). In 1985, a high spray year because of a special congressional appropriation to control noxious weeds on Federal land, 7,000 pounds of herbicide (a.i.) were sprayed. In 1986, this figure dropped to about 4,000 pounds. In 1987, approximately 7,400 pounds of herbicide were applied. Typically, less than 10,000 acres are sprayed.

In 1987, the herbicide 2,4-D comprised approximately 70 percent of the herbicide used to control noxious weeds in the Northern Region. Picloram use was approximately 25 percent of the total. Use of dicamba has been dropping in the past several years and now comprises about 1 percent of the total. Annual use of less than 50 pounds of glyphosate is expected in the future because of the nonselective nature of this herbicide. On some forests, tebuthiuron pellets have been substituted for picloram pellets which have been withdrawn from the market.

Herbicide application rates are expressed on an active ingredient basis as pounds per acre (lb/ac) or kilograms per hectare (kg/ha). Application rates depend primarily on the species of weed being controlled and, to a lesser extent, on site-specific variables such as soil type, stage of growth, or moisture conditions. Table 2-1 provides typical active ingredient application rates for various herbicides and mixtures of herbicides. These application rates will be used in the risk analyses with allowances for application errors discussed in Section 2.2.

In some cases, the application rates in the Northern Region may differ from those provided in Table 2-1. For example, picloram can provide 99 to 100 percent control of knapweed when applied at only one-quarter pound per acre. The use of the application rates on Table 2-1 plus allowances for application errors will, in itself, overestimate potential impacts of many spray projects. When comparing model projects to site-specific projects, daily dosage estimates may be adjusted to account for differences in application rates.

In large areas with spotty infestations of target plants, the term treatment project must be applied somewhat arbitrarily. In this analysis, treatment sites within one-half mile of each other are grouped together as one project. Sites that are greater than one-half mile from the next nearest site are considered to be separate projects.

Table 2-1. Pesticide application rates.

Herbicide	Application rate lbs/ac	Application rate kg/ha
2,4-D	2	2.2
Glyphosate	1	1.1
Picloram	1	1.1
2,4-D/ Picloram[1]	0.75 0.25	0.8 0.3
2,4-D/ Dicamba[2]	0.75 0.25	0.8 0.3
Amitrole[3]	1	1.1
Atrazine	1	1.1
Hexazinone	1	1.1
Tebuthiuron	1	1.1
Dicamba	1	1.1

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[1] Applied as a tank mix.

[2] Applied as a tank mix or a commercial product.

[3] Amitrole, atrazine, and hexazinone are not currently used for weed control in the Northern Region. These herbicides have been used in other regions of the Forest Service.

In discussing a project, a distinction is made between gross and net area. Net area is the area actually treated with herbicide. Gross area is the area inside the smallest perimeter incorporating all the treatment sites and includes both the treated and untreated area. The gross area can often be 10 times or more than the net area treated.

Treatment projects are divided into one of the three following categories based on location: open-range and forest lands, road rights-of-way (ROW), and riparian projects. General area NFS lands are used for grazing, harvest of wood products, wildlife habitat, recreation, and other purposes. Right-of-way projects involve treating strips of land immediately adjacent to roads including areas in recreational sites. Riparian projects involve treating areas in which at least part of the herbicide is applied in riparian zones which are typically adjacent to flowing or standing water. Road ROW and riparian projects are designed primarily to slow the spread of noxious weeds or poisonous plants by vehicles or water. Most herbicide spraying in riparian areas occurs with the spraying of road ROW which often parallel stream channels.

The five model projects which form the basis of these risk analyses include four project types within the open-range/forest category (small, mid-sized, and two large projects (ground application and aerial application)). The fifth model project is a combination travelway and riparian project. The critical elements of these projects are defined such that the apparent risk of these projects is greatly increased. The descriptions of these model projects will indicate the assumptions used to develop these scenarios.

Although no actual project will be identical to any one of the models, the risks involved in any actual project will typically be less than the risks assumed in the model project category to which it would be assigned. Although these model projects are based on assumptions that overestimate risk from most, if not all, actual projects, it cannot be assumed that every project in the future will have less risk than a corresponding model project. Proposed projects will be reviewed to determine whether the project represents a higher risk to affected populations based on proximity to population centers, size of project, number of contiguous projects, and the amount or type of herbicide used.

2.1.1 Small Model Project

The small model project is assumed to involve herbicide applications to 1 net acre (.4 hectare) spread over a 10 acre (4 hectare) gross area. The plots are presumed to be sprayed in an area adjacent to a section of private land containing a residence with four inhabitants. The residence is assumed to be approximately 220 yards (200 meters) from the project. In addition, the residence is assumed to be directly downwind from the herbicide application.

The assumption that land in private ownership is in close proximity to a small project is conservative since National Forests typically comprise large unbroken expanses of Federal land with minor amounts of private inholdings. In those areas where National Forest System and private lands are interspersed in a checkerboard fashion, the private holdings are most often commercial timberlands with no residental populations. In the eastern Montana and North Dakota National Grasslands, the inholdings would comprise parts of large, sparsely populated ranches often involving as many as 10 to 20 sections (square miles) of land.

The treatment areas are assumed to be sprayed by two individuals with backpack sprayers. Each applicator spends 2 hours on a small spray project. These two workers are each assumed to work 6 hours per day and treat not more than three one-acre projects in one day.

2.1.2 Mid-sized Model Project

The mid-sized model project on general areas is assumed to involve herbicide application to approximately 40 acres (16 hectares) of target plants spread over a 200-acre (80-hectare) plot. As with the small project, the spray area is assumed to be located adjacent to a residence in an privately owned section. As with the smaller projects, the closest border of mid-sized project is assumed to be within 220 yards (200 m) upwind of the residence.

Treatment of each mid-sized project is assumed to be accomplished by four applicators with backpack sprayers. Each applicator spends 6 days on this type of project.

2.1.3 Large, Ground Application Model Project

In a typical year, the National Forest System will spray relatively few areas with continuous extensive infestations of noxious weeds. This risk analysis assumes a large project of 500 acres (200 hectares). The 500-acre plot is assumed to be located within 220 yards (200 meters) of a residence.

The close proximity of a large spraying project to neighboring residences would be highly unusual. Such projects are typically located in the interior of large tracts of National Forest System land. The configuration of private residences and sprayed areas again presents a conservative basis for assessing risk of spray operations to the general public.

These large projects would typically be sprayed with vehicle-mounted spray equipment. Edges and areas of rough terrain may be sprayed with backpack units. Because worker exposure is higher with backpack sprayers, it is assumed that 40 hectares (100 acres) are sprayed with these spray units and the remainder with vehicle-mounted spray equipment.

2.1.4 Right-of-Way and Riparian Model Project

Invading plants often spread initially along roads, trails, and streams which serve as transportation corridors for seeds. As a consequence, some travelways and areas near water will be treated in order to remove seed sources and reduce poisonous plant hazards to humans and animals.

The ROW model project is assumed to involve 10 miles of roadside (5 miles of road treated on both sides) sprayed in a 20-foot swath. The spray area would total about 24 acres or 9.8 hectares. It is assumed that two residences are located within 225 feet (60 meters) of the spray zone at either end of the spray corridor.

The number of residents hypothetically impacted by this model treatment area is higher than would normally be impacted since most NFS roads transect land that is administered by the Forest Service and contains little or no human habitation.

A small stream with a flow rate of approximately 1 cubic foot per second (cfs) is assumed to parallel the road approximately 15 feet from the closest spray point. Immediately downstream of the spray area, the small stream is assumed to flow into a 15 cfs stream that is capable of supporting a fishery.

Road rights-of-way are often treated with truck-mounted equipment with a boom extension or side nozzles, usually capable of reaching 12 feet from the side of the truck. Often the entire 20-foot (6-meter) roadside swath can be reached in one pass with a combination of spray nozzles mounted off the front bumper plus the 12-foot extension boom or side nozzles. On rough terrain, the truck must remain on the roadway. In this case, the 12 feet nearest the road would be covered by the extension boom. The remaining area would be spot-treated with a hand-held spray gun connected by a hose to the vehicle-mounted spray rig. Because worker exposure could be higher under the second application routine, in keeping with the conservative nature of these analyses, it is assumed that all travelway and riparian treatment areas involved a combination of boom spray and hand-held nozzle application methods.

2.1.5 Large, Aerial Application Model Project

The scattered nature of most target plant infestations generally prevents the effective aerial application of herbicides. Occasionally large areas of heavy weed infestation could be treated aerially.

This analysis assumes that a large 120-acre (49-ha) area is treated aerially to control target plants. Once again, it would be unusual for residences to be in the vicinity of these spray projects. This analysis assumes a residence within 220 yards (200 meters) of the spray project.

A small stream with a flow rate of approximately 1 cubic foot per second is assumed to parallel the treatment area approximately 55 yards (50 meters) from the closest spray point. Immediately downstream of the spray area, the small stream is assumed to flow into a 15 cfs stream that is capable of supporting a fishery.

2.2 ERRORS AND MISAPPLICATIONS ASSOCIATED WITH HERBICIDE SPRAY PROJECTS

These analyses assume that the level of exposure and dose to both workers and the general public is directly related to the amount of herbicide applied per unit area. Deviations from the prescribed application rates can be affected by several factors including:

- Errors of measurement during manufacture and formulation.
- Errors of measurement and calibration during field mixing.
- Excessive swath overlap during application.

The extent and probability of these types of errors affecting application rates are discussed in this section. In addition to these factors, two other types of misapplication are discussed:

- Use of a herbicide not scheduled for a particular area.
- Treatment of an area not scheduled for treatment.

In addition to these operation errors, the impacts of major accidents such as truck spills onto land or into domestic water sources are discussed in Section 3.

2.2.1 Errors of Measurement During Manufacturing

Pesticide manufacturers and formulators are required by U.S. EPA to maintain the concentration of pesticide in a product to within 4 percent of the stated concentration. Although the true concentration of various samples of a pesticide product would probably cluster about the labeled concentration, these risk analyses assume that the true concentration of the herbicide is always 4 percent greater than the labeled concentration. This assumption will increase the estimated public health and environmental impacts of herbicide treatment programs.

2.2.2 Errors of Measurement in the Field

Most herbicide formulations require additional dilution for field applications. Errors can occur due to improper calibration of metering equipment, miscounting, or misinterpretation of instructions for use of measuring instruments. Again it is expected that the actual diluted concentration would cluster about the appropriate dilution rate and no accumulative error would occur. However, this analysis assumes that 10 percent of the mixtures for field applications are mixed such that the herbicide concentration is 10 percent higher than prescribed. In addition, the analysis assumes that 1 percent of the field mixtures are mixed tenfold overstrength.

2.2.3 Excess Swath Overlap During Application

It is assumed that 5 percent of the land sprayed on any individual project is sprayed twice due to swath overlap.

The total impact of the various errors can be calculated for two different scenarios. In the first scenario the pesticide is assumed to be formulated 4 percent overstrength; the field mixture is assumed to be mixed 10 percent overstrength 10 percent of the time and is assumed to be mixed properly 90 percent of the time; and, finally, 5 percent of the spray area is assumed to be sprayed twice due to swath overlap. In this case, the assumed application rate on an area prescribed at 1.1 kg/ha (1.0 lb/ac) would be 1.2 kg/ha (1.1 kg/ha x 1.04 (formulation error) x 1.10 (10 percent mixing error) x 0.10 (10 percent probability factor) x 1.05 (5 percent double swath)) + (1.1 kg/ha x 1.04 (formulation error) x 0.90 (90 percent probability factors) x 1.05 (5 percent double swath)). The first scenario will be referred to henceforth as the minor mixing error scenario.

In the second scenario, the pesticide is assumed to be formulated 4 percent overstrength; the field mixture is assumed to be mixed 10 percent overstrength 10 percent of the time, tenfold overstrength 1 percent of the time, and mixed properly 89 percent of the time; and, finally, 5 percent of the area is assumed to be sprayed twice due to swath overlap. In this case, the assumed application rate on an area prescribed at 1.1 kg/ha (1.0 lb/ac) would be 1.3 kg/ha ((1.1 kg/ha x 1.04 (formulation error) x 1.10 (10 percent mixing error) x 0.10 (10 percent probability factor) x 1.05 (5 percent double swath)) + (1.1 kg/ha x 1.04 x 10 (tenfold overmix) x 0.01 (1 percent probability factor) x 1.05) + (1.1 kg/ha x 1.04 x 0.89 (89 percent probability factor) x 1.05). The second scenario will be referred to henceforth as the major mixing error scenario.

Using the assumptions of the major mixing error and minor mixing error scenarios, the assumed application rates also can be calculated for the other typical prescriptions as shown on Table 2-1.

2.2.4 Use of a Herbicide not Scheduled for a Particular Area

The USDA Forest Service in the Northern Region uses primarily 2,4-D, dicamba, and picloram for the chemical control of noxious weeds. In many cases, 2,4-D will be applied as a mixture with one of the other two herbicides. The herbicide use pattern at the National Forest or Ranger District level is even simpler and is often limited to one or two of the above herbicides depending on the weed infestations of the local area. Thus, the possibility of applying the wrong herbicide to a location is small and generally of little consequence to these analyses.

More importantly, the herbicides of major use do not differ significantly in most areas of human health and environmental impact. Where differences do exist, this risk analysis analyzes the most significant impacts in a particular location from herbicide use. For example, this analysis examines the impact of picloram use in riparian habitats although label directions prohibit many uses of picloram near water. This herbicide is relatively mobile in soil/aquatic environments and weak evidence of carcinogenic potential for this herbicide exists.

Again it is emphasized that assumptions concerning use patterns, e.g., picloram by streamside, are made solely for the purpose of establishing a worst-case scenario for these risk analyses and are not indicative of Forest Service herbicide-use policies.

2.2.5 Treatment of an Area not Scheduled for Treatment

Generally speaking, the model application projects outlined in Section 1 incorporate conservative (worst case) assumptions to assess potential impacts. The application of herbicide to an area not scheduled for treatment would likely result in impacts to humans less severe than those analyzed within the framework of the model projects.

The isolated location of most NFS land insures large untreated zones between treated areas and inhabited areas. The possibility of mistaken treatment of areas very close to human habitation is accounted for in the assumption that small, mid-sized, and large projects are within 220 yards (200 meters) of residences. It is assumed that right-of-way and riparian projects are sprayed within 60 meters of residences.

The effects of a major accidental spill of herbicide on sensitive areas is discussed in Section 3.

2.3 AFFECTED POPULATIONS

The population that could be affected through exposure to herbicides used to treat target species can be divided into two sets. The first set includes workers involved in the application of herbicides: truck drivers, pilots, mixer/loaders, handspray applicators, and supervisors. The second set is composed of the general public subject to nonoccupational exposure. This group includes residents in the vicinity of sprayed areas, visitors to sprayed areas, and consumers of products potentially contaminated by herbicides.

2.3.1 Small Projects: Affected Populations

As discussed in Section 2.1.1, two applicators with backpack sprayers would spend approximately 2 hours each in spraying a small project. This pair of applicators is presumed to cover three small projects per six-hour application day. No other workers or supervisors are assumed to be directly involved in the handling and application of these herbicides.

As discussed in Section 2.1.1, it is assumed that there is one residence with four inhabitants 220 yards (200 meters) directly downwind of each small project. The residents are assumed to include two adults (154 pounds or 70 kg average weight), one adolescent (88 pounds or 40 kg), and a 2-year-old (26 pounds or 12 kg). These inhabitants are assumed to be outside during the entire spray period and thus exposed directly to spray drift.

The residents are also assumed to have a vegetable garden adjacent to their house and directly downwind of the spray zone. The residents are assumed to slaughter a steer for personal consumption immediately after it grazed on herbicide-treated forage for a sufficient time to allow maximum accumulation of herbicide in body tissues. This beef provides the sole source of meat for these inhabitants for 140 days.

As is demonstrated in Section 2.4, none of these herbicides bioaccumulate to any extent in mammalian, avian, or aquatic species and are rapidly eliminated after ingestion. Impacts on animals are quite transient and a secondary human dose of herbicides could only occur if the animals are slaughtered shortly after exposure. The big game hunting season normally begins 1 to 2 months after treatment. Therefore, because of the small percentage of NFS land being treated, the wide-ranging habits of these animals, and the time intervals between treatments and the hunting season, the impacts from eating wild game which may have grazed on herbicide treated plants are negligible by comparison to the worst-case impacts from beef (as demonstrated in Section 2.4). Because the worst-case scenario assumes that beef that is maximally contaminated with herbicide is the sole source of meat for the residents, any substitution of another meat source would lessen the dose.

In addition to inhabitants near the sprayed area, visitors are assumed on the sprayed area. National Forest System records indicate that the 10.1 million hectares (25 million acres) of Northern Region National Forest and Grasslands experience approximately 11.4 million visitor-days/year (a visitor day is considered to be 12 hours spent on forest land). Therefore, on average, Forest Service land in Region 1 receives about 1.25 visitor-day per hectare per year or one-half visitor-day per acre per year assuming a random distribution of forest visitors. Since most spraying will occur in areas with virtually no visitation at any time, the random distribution assumption will result in a high estimate of visitors to treated areas. Further, this risk analysis assumes that the half day of visitation to the small project occurs immediately after spraying.

National Forest System recreation records indicate that less than 1.0 percent of Forest visitors gather edible wild plant foods. These risk analyses assume that 1.0 percent of the visitors collect 0.5 pound of edibles from the treated areas. This is an overestimate because prime huckleberry patches or other berry fields are generally not located in habitats that are targeted for herbicide use. In addition, weeds are normally treated several weeks prior to the period for ripe berries, and any direct spraying of this vegetation would brown the leaves and prevent fruit development.

2.3.2 Mid-sized Projects: Affected Populations

As discussed in Section 2.1.2, treatment of a mid-sized project is assumed to require four applicators with backpack sprayers 6 days each on this project. No other workers or supervisors are assumed to be directly involved in handling or application of herbicides.

The similar assumptions regarding proximity and number of residents are made for both small and mid-sized projects. Specifically, a residence with four inhabitants is assumed on the adjacent section of land 200 meters directly downwind of the treatment area. The assumptions made for small projects regarding garden location and consumption of beef by residents are also made for mid-sized projects.

As with the small projects, a visitor rate of 0.5 visitor-day/acre is used for mid-sized projects. Visitors are assumed to be on-site shortly after treatment and less than 1.0 percent of visitors gather wild food from the site.

2.3.3 Large-sized Ground Application Projects: Affected Populations

Large, continuous infestations would be treated with vehicle-mounted spray rigs. Rough terrain, treatment block edges, and other hard to reach places would be treated with various hand-held applicators. This risk analysis, assumes that 80 percent (400 acres) of the project area would be treated with vehicle-mounted spray equipment and that 20 percent (100 acres) of each project area would be treated with various hand-held application devices. The estimated portion sprayed by hand is probably high and serves to increase apparent worker exposure from these projects because worker exposure is higher from hand applications than from any other method of spray application. It is assumed to require ten days of vehicle spraying per project by a truck driver who does his own mixing and loading. Six workers with backpack sprayers spray the remaining 20 percent in a total of 60 worker days per project. One supervisor directs all worker activities.

As described in Section 2.1.3, the large project includes a residence situated 220 yards (200 meters) downwind from each project area. Similar assumptions regarding number of inhabitants, location of gardens, consumption of beef, etc., are made for large projects as for the small and mid-sized projects.

Visitor use is based on one visitor per 2 acres and 1 percent of the visitors are assumed to gather wild edible food.

2.3.4 Right-of-way and Riparian Projects: Affected Populations

As discussed in Section 2.1.4, it is assumed that ROW and riparian projects would be treated using a combination of boom sprayer and hand-held nozzle or backpack sprayers. This application would require one truck driver and one hand nozzle operator. Approximately one work day would be spent by these two people on each project treatment area.

The 10 miles (16 kilometers) of roadside treatment in each model project area are assumed to be equally distributed on each side of the road. Two residences with four inhabitants each are assumed to be located on either end of the treatment zone approximately 200 feet (60 meters) downwind. Inhabitant ages and weights are as presented for small projects in Section 2.3.1. Each resident is assumed to be outside during the treatment. In addition, one 12-year old child weighing 88 pounds (40 kg) is assumed to be attracted by the treatment activity and to be three feet downwind of the treatment zone during the treatment operation. Two visitors are assumed to walk the length of the treated area daily.

The inhabitants in the vicinity of the treatment area are assumed to have a vegetable garden adjacent to their residence. The family also slaughters and consumes a steer which has grazed on contaminated forage. A fisherman is assumed to catch ten 8-ounce fish from the larger streams downstream of the spray zone.

2.3.5 Aerial Application Projects: Affected Populations

Relatively large crews are typically required for aerial spray projects. These include a pilot, a mixer/loader, Forest Service supervisors, observers, and flagmen. This analysis assumes that the 120-acre spray project could be sprayed in a morning. Meteorologic conditions are typically more appropriate for aerial spraying in the morning (lower wind speeds and temperatures).

As described in Section 2.1.4, the project includes a residence 220 yards (200 meters) downwind of the project. Similar assumptions regarding number of inhabitants, location of gardens, consumption of beef, etc., are made for large, aerial projects as for small and mid-sized projects.

Visitor use is based on one visitor per 2 acres and 1 percent of the visitors are assumed to gather wild food.

2.4 EXPOSURE LEVELS FOR AFFECTED POPULATIONS

This section will provide data on the level of exposure and subsequent dose to workers involved in herbicide application as well as to the general public.

An important distinction should be made between exposure to herbicides and subsequent dosage. Exposure refers to the contact or potential contact between the chemical compound and the surface of the organism prior to incorporation of the chemical into cells or organs. The dose refers to the portion of the substance that is taken into the organism as a result of exposure. This distinction is made for several reasons, e.g., exposure to herbicides during application is often a function of physical variables such as spray equipment, wind speed, height of application, and concentration of herbicide applied. Thus, the dermal exposure of a worker using a backpack sprayer will be similar whether using 2,4-D, dicamba, glyphosate, or any other herbicide as long as all other variables are held constant.

The dose or amount absorbed from this exposure will often depend on chemical characteristics of the herbicide. For example, the dermal dose will be a function of the nature of the chemical and its interaction with skin. The actual dose is specific to each herbicide although certain generalities on rate of absorption are possible and will be developed in this section.

A distinction should also be noted between the terms dose and dosage. The dose is the quantity of the substance taken in (typically in weight units such as milligrams). Dosage is expressed as the amount of substance per unit of body weight (milligram per kilogram or mg/kg).

As noted in Section 1, the herbicides dicamba, 2,4-D, and picloram are expected to account for over 97 percent of the chemicals used to control target species. These analyses provide exposure and dosage data for these herbicides as well as amitrole, hexazinone, glyphosate, tebuthiuron, and atrazine.

2.4.1 Introduction to Worker Exposure and Dosage

Exposure and dosage factors for workers involved in applying herbicides of interest in this study are based primarily on studies of workers applying 2,4-D, dichlorprop, and picloram (Lavy et al. 1982 and 1984 and Nash et al. 1982).

These studies analyzed the urine of workers for the herbicides of interest as an indication of worker dose from all routes (dermal, respiratory, and oral). These studies also provided data on the amount of herbicide applied by these workers during the study period which allowed normalization of the data on a "per kilogram applied (or mixed)" basis. Other studies that did not provide such complete information are cited below as necessary to extend our understanding of worker dosage.

Table 2-2 summarizes the results of the Lavy studies and the Nash study. For each worker category in Table 2-2, two dosage levels are provided. The first is the average dosage of all workers studied in the category. The second or high dosage was calculated by adding two standard deviations to the average dosage. The high dose was calculated using common statistical techniques to indicate the dose level that would be higher than about 98 percent of worker doses under similar conditions (i.e., similar protective clothing, application techniques, etc.).

Lavy et al. (1984) also measured picloram dosage to workers who applied a combination of 2,4-D and picloram. Although the application methods differed from those employed for noxious weed control, the Lavy study indicates that when applied under comparable circumstances the picloram dosage will be 5 to 12 times lower than the 2,4-D dose. This difference is in large part a function of the differences in the rate at which 2,4-D and picloram are absorbed through the skin.

In addition to the Lavy studies and the Nash study, Draper and Street (1982) measured via urinalysis the worker dose during applications of a mixture of 2,4-D and dicamba. This study alone does not indicate how dicamba dose relates to the application rate. However, because the workers applied a mixture of 2,4-D and dicamba, the study indicates that dicamba dose to workers will not exceed 2,4-D dose under similar work conditions. Thus, data on 2,4-D from the Lavy et al. and Nash et al. studies can be extrapolated to dicamba.

Several herbicides of interest in these risk analyses are not included in studies by Lavy et al. and by Nash et al. In order to estimate doses of these herbicides several factors impacting worker dose should be reviewed. Worker dose involves three exposure pathways: the dermal adsorption of herbicide drift impacting the skin, inhalation of herbicide mist, and oral doses. By measuring the quantities of herbicide impinging respirator filters, several studies including Lavy et al. (1982 and 1980b) have demonstrated that the inhalation exposure is typically negligible (less than 1 percent of the amount from other routes).

Table 2-2. Summary of 2,4-D dosage data developed from Nash et al. (1982), Lavy et al. (1982), and Lavy et al. (1984)[1].

	Nash (1982)		Lavy (1982)	
	Average	High	Average	High
Truck/tractor driver	$0.09\text{x}10^{-3}$	$0.35\text{x}10^{-3}$		
Mixer/loader (ground)	$0.402\text{x}10^{-3}$	$1.04\text{x}10^{-3}$		
Mixer/loader/driver	$0.85\text{x}10^{-3}$	$3.2\text{x}10^{-3}$		
Pilot			$0.16\text{x}10^{-3}$	$0.64\text{x}10^{-3}$
Mechanic			$0.047\text{x}10^{-3}$	$0.16\text{x}10^{-3}$
Mixer/loader (aerial)			$0.184\text{x}10^{-3}$	$0.48\text{x}10^{-3}$
Supervisor			$0.024\text{x}10^{-3}$	$0.11\text{x}10^{-3}$
Observer			$0.005\text{x}10^{-3}$	$0.013\text{x}10^{-3}$
Backpack workers (from Lavy et al., 1984)			0.086	0.176

[1] All data are in milligrams of herbicide absorbed per kilogram of body weight per kilogram of herbicide mixed and/or applied. Please note that these data cannot be used unless the amount of herbicide that the worker mixes and/or applies in a day is estimated. See text for discussion. Section 1.9 provides instructions on converting exponential notation.

As noted above, the difference in picloram and 2,4-D dosages is probably a function of differences in dermal absorption rate. Nolan et al. (1984) has shown dermal absorption of picloram to be less than 0.2 percent whereas the measured dermal absorption rate for 2,4-D ranges from 7 percent to 28 percent depending on the body part exposed (Feldman and Maibach 1974 and Maibach et al. 1971).

The difference in 2,4-D and picloram worker dosages is not as great as the difference in dermal absorption between these herbicides. In addition, those workers with the highest doses showed less difference in their picloram and 2,4-D doses than did workers with average doses. Both of these observations indicate that workers are receiving part of their doses orally. The more careless the worker's personal habits the higher his oral exposure and dose and the smaller the difference in 2,4-D and picloram doses.

In summary, large differences between herbicides in their dermal absorption rates will result in some differences in worker dosages under otherwise similar conditions. However, because of the possibility of oral exposure (wiping mouth with hands, eating with contaminated hands, etc.), worker dosage will not be solely a function of dermal absorption. In the case of picloram with a dermal absorption rate of 0.2 percent, worker dosage rates will be assumed to be one-fifth of 2,4-D rates based on the comparative worker doses observed in the Lavy study (1984).

Prediction of worker dosage from the application of the other herbicides would be facilitated by data on dermal absorption rates based on human tests. Unfortunately dermal absorption rate data for humans for the herbicides amitrole, atrazine, glyphosate, tebuthiuron, and hexazinone are not available. However, data from animal studies of the dermal absorption of these herbicides define the outer limits of dermal absorption in humans.

Tests of glyphosate on monkeys have shown dermal absorption of 2 percent of applied material (Peterson 1983). Tests of amitrole on rats indicate that amitrole has a dermal penetration potential of 0.1 percent (U.S. EPA 1985b). Tests of atrazine on rats have shown dermal absorption rates of 18 percent after 12 hours (Ballantine 1985). In these cases, the figures represent high estimates of dermal absorption in man based on an extensive literature review of dermal absorption including a review of interspecies comparative studies with a variety of compounds (Levin et al. 1984). Data reviewed indicate that dermal absorption rates in rats are typically several times higher than dermal absorption rates in man. Dermal absorption rates for monkeys more closely approximate those of man but once again overestimate rates in man. These species differences are likely a function of skin thickness, number of hair follicles, and other factors.

Based on a similarity of dermal absorption of amitrole and picloram, worker dosage rates for picloram will be used for amitrole. Dermal absorption rates of glyphosate, atrazine, and 2,4-D are in the same range, and the same worker dosage rates will be used for all three herbicides. In addition, based on the work by Draper and Street (1982) discussed above, dicamba and 2,4-D worker dosage factors are assumed to be similar.

Quantitative dermal absorption rates for hexazinone are not available for any animal species. However, an estimate of dermal absorption rate of hexazinone can be made by comparing the acute dermal and the acute oral toxicity data for these herbicides. Since only a fraction of the chemical to which the animal is dermally exposed is absorbed into the body, dermal toxicity should be less than oral toxicity.

A review of mammalian test data for the compound hexazinone as contained in Agriculture Handbook No. 633 (USDA, Forest Service 1984) indicates that hexazinone has no lethal effects with dermal exposures as high as 6,000 mg/kg in rabbits. As discussed above, a comparison of dermal absorption of various chemicals by several mammalian species (rats, monkeys, rabbits, hairless pigs) shows the highest dermal absorption in rabbits (see Levin et al. 1984). Dermal exposure test results with hexazinone indicate that it is virtually impossible to induce a lethal response in mammals through dermal exposure.

Oral toxicity of hexazinone is also relatively low, although lethal doses are possible. An oral LD_{50} (lethal dose to 50 percent of animals treated) of 860 mg/kg is indicated as a conservative value (USDA, Forest Service 1984). Because dermal exposures as high as 6,000 mg/kg are not lethal, it is obvious that relatively little hexazinone is absorbed through skin. A dermal absorption rate comparable to glyphosate or 2,4-D is very likely applicable for hexazinone. However, because specific dermal absorption rates for animals or humans are not available, the worker dose rates for 2,4-D are doubled to estimate the worker dose for hexazinone.

Tebuthiuron is unique in that the Forest Service will only apply this compound as a granular formulation containing 1 or 5 percent active ingredients in clay pellets. Since these clay pellets do not drift or adhere to skin there is little possibility of measurable worker dose. Possible inhalation of small amounts of dust provide the only hazard. Tebuthiuron worker dose is thus assumed to be 1 percent of the 2,4-D dose.

Table 2-3 provides the worker dose factors used in these analyses for all worker functions except backpack sprayers which are discussed separately below. These dose factors are expressed as the milligrams of herbicide absorbed per kilogram of worker body weight per kilogram of herbicide applied. Therefore, in order to calculate worker dose, the quantity of herbicide applied daily must be calculated (in kilograms of active ingredient) and multiplied against the factors in Table 2-3.

The backpacker dosage factors cited in Table 2-2 are based on a study by Lavy et al. (1984) of workers spraying bushes 5 to 15 feet high. As a result, blowback, drip, and dermal contact with sprayed vegetation resulted in herbicide exposure that was much higher than that encountered by workers spraying noxious weeds 3 feet tall or less. In addition, these workers often wore little in the way of protective clothing, although attempts to increase worker protection were overwhelmed by the extreme conditions under which the herbicide was applied.

Under conditions more typical of noxious weed spraying, research has shown that protective clothing can substantially reduce worker exposure. For example, in right-of-way spraying, doses of spray gun applicators wearing clean coveralls and gloves were reduced by 68 percent compared to the doses they got without this protection (Libich et al. 1984). During insecticide applications to orchards, mixers reduced their exposure by 35 percent and sprayers reduced their exposure by 49 percent by wearing coveralls (Davies et al. 1982). Putnam and coworkers found that nitrofen applicators and mixer/loaders wearing protective clothing reduced their exposure by 94 to 99 percent compared to the doses experienced without protection (Waldron 1985).

As discussed above, most application exposure to herbicides is dermal, not respiratory (Kolomodin-Hedman et al. 1983). Thus, the use of respirators is generally unnecessary, except where concentrated mixtures are used in enclosed spaces. The hands are the site of the greatest potential herbicide exposure, and rubber gloves are generally quite effective in preventing exposure to hands (Putnam et al. 1983).

Table 2-4 presents a range of backpack applicator dosage factors. The recommended protection values for both average and high dosage levels assume that protective clothing will reduce worker dose by 68 percent from the low protection values based on the Lavy studies. Recommended protection values are only provided for workers applying tebuthiuron pellets since minimal protection is required for pellet applications.

Table 2-3. Worker dosage factors.[1/][2/][3/]

	Amitrole Picloram		Atrazine, Dicamba, 2,4-D & Glyphosate		Hexazinone	
	Average	High	Average	High	Average	High
Truck/tractor driver (including mixing and loading)	1.7×10^{-4}	6.4×10^{-4}	8.5×10^{-4}	3.2×10^{-3}	1.7×10^{-3}	6.4×10^{-3}
Mixer/loader (aerial)	3.6×10^{-5}	9.6×10^{-5}	1.8×10^{-4}	4.8×10^{-4}	3.6×10^{-4}	9.6×10^{-3}
Pilot	3.2×10^{-5}	1.3×10^{-4}	1.6×10^{-4}	6.4×10^{-4}	3.2×10^{-4}	1.3×10^{-3}
Mechanic (aircraft)	9.4×10^{-6}	3.1×10^{-5}	4.7×10^{-5}	1.6×10^{-4}	9.4×10^{-5}	3.2×10^{-4}
Supervisor	4.8×10^{-6}	2.2×10^{-5}	2.4×10^{-5}	1.1×10^{-4}	4.8×10^{-5}	2.2×10^{-4}
Observer	1.0×10^{-6}	2.6×10^{-6}	5.0×10^{-6}	1.3×10^{-5}	1.0×10^{-5}	2.6×10^{-5}

[1/] All values are expressed in milligrams of herbicide absorbed per kilogram of body weight per kilogram of herbicide mixed and/or applied. In order to calculate worker dosage, the appropriate factor from this table must be multiplied by the amount of herbicide (kilograms of active ingredient) applied daily by each worker.

[2/] Except backpack spray and pellet applicators.

[3/] Tebuthiuron worker dosage factors associated with aerial application are assumed to be 1 percent of 2,4-D factors.

Table 2-4. Worker dosage factors[1] for backpack spray applicators and pellet applicators.

	Recommended Protection		Low Protection	
	Average	High	Average	High
Amitrole	5.6×10^{-3}	1.1×10^{-2}	1.7×10^{-2}	3.5×10^{-2}
Atrazine	2.8×10^{-2}	5.6×10^{-2}	8.6×10^{-2}	1.76×10^{-1}
2,4-D	2.8×10^{-2}	5.6×10^{-2}	8.6×10^{-2}	1.76×10^{-1}
Dicamba	2.8×10^{-2}	5.6×10^{-2}	8.6×10^{-2}	1.76×10^{-1}
Glyphosate	2.8×10^{-2}	5.6×10^{-2}	8.6×10^{-2}	1.76×10^{-1}
Hexazinone	5.6×10^{-2}	1.1×10^{-1}	1.7×10^{-1}	3.5×10^{-1}
Picloram	5.6×10^{-3}	1.1×10^{-2}	1.7×10^{-2}	3.5×10^{-2}
Tebuthiuron	8.6×10^{-4}	1.76×10^{-3}	NA[2]	NA[2]

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[1] All values are expressed in milligrams of herbicide absorbed per kilogram of body weight per kilogram of herbicide applied. In order to calculate worker dosage, the appropriate factor from this table must be multiplied by the amount of herbicide (kg of a.i.) applied daily by each worker.

[2] See discussion in text.

2.4.2 Introduction to General Public Exposure and Dose from Ground Spray Equipment

Off-target drift of herbicide during herbicide applications represents one of several ways in which persons in the vicinity of treated areas can be exposed to herbicides.

Several investigators (Yates et al. 1978; Maybank et al. 1977; USDA Forest Service 1984a) have studied drift of herbicides from ground equipment as well as from aircraft. Yates and his coworkers provide the most complete study of drift from ground-rig applications over relatively long distances (up to 0.6 mile or 1,000 meters). Maybank and his coworkers provide more complete data concerning deposition on target and the deposition and drift of herbicide within short distances off target. Both types of data will be useful in determining the impacts of treatment under different application scenarios outlined in Section 2.1.

The USDA Forest Service has developed several models to predict deposition and drift during aerial spray projects. This analysis uses the Forest Service Cramer-Barry Grim (FSCBG) Model to predict drift from the 120-acre model aerial project (USDA, Forest Service 1984a).

In determining rates of drift from ground application, the highest rates of drift found in tests of ground equipment by Yates and his coworkers or by Maybank and his coworkers are assumed to occur at all times during ground application in these analyses. These drift data very probably overestimate drift from typical ground application since other tests have shown drift as much as 100 times lower. In addition, the drift rates used here were based on drift from tractor- or truck-mounted spray equipment employing high-pressure spray booms and treating over 3 feet above the ground. Although drift from lower pressure backpack spray equipment is expected to be much less than the rates extrapolated from vehicle-mounted equipment, in the absence of extensive data on backpack sprayers, the drift rates from vehicles were applied across the spectrum of ground equipment.

Table 2-5 presents data extracted from Yates et al. (1978) on the deposition of drift onto downwind mylar sheets. Data for 10-meter-wide swaths are based on experimental results. Data from 100-meter-wide swaths were calculated by Yates from the 10-meter results. Data on Table 2-5 are expressed in a form such that drift deposition at a specified distance is given in mg/m^2 (milligrams per square meter) when the appropriate factor is multiplied by the application rate in kilograms per hectare (kg/ha). For example, at 100 meters downwind from a 10-meter-wide application at 1.3 kg/ha, the drift deposition would be 0.0156 mg/m^2 (.012 $mg\text{-}ha/kg\text{-}m^2$ x 1.3 kg/ha).

Table 2-5. Highest drift-deposition levels collected on mylar sheets at specified distances from ground application spray projects (Yates et al. 1978).

Distance (meters)	Drift deposition from a 10-meter-wide spray swath[1]	Drift deposition from a 100-meter-wide spray swath[1]
60	0.025	0.095
100	0.012	0.064
200	0.0048	0.036
300	0.0024	0.024
400	0.0017	0.017
500	0.0012	0.012
600	0.0009	0.010
700	0.0008	0.008
800	0.0007	0.007
900	0.0006	0.006
1,000	0.0005	0.005
1,100	-	0.004
1,200	-	0.004
1,300	-	0.003
1,400	-	0.003
1,500	-	0.002
1,600	-	0.002

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[1] Drift deposition is presented as milligrams per square meter when the application rate in kg/ha is multiplied by the appropriate factor from this table. Example: 1.3 kg/ha x .036 mg-ha/kg-m^2 = .0468 mg/m^2. (Drift deposition 200 meters from a 100-meter-wide swath sprayed at 1.3 kg/ha.)

Table 2-6 presents drift deposition data from Yates et al. (1978) for wheat plants located downwind of 10-meter-wide swaths (experimental data) or 100-meter swaths (calculated). Since wheat has a very high surface to mass ratio, this data will be a worst-case indication of concentration on leafy vegetables in gardens in the drift zone. Drift deposition on vegetation at 200 meters from a 10-meter-wide swath treated at 1.3 kg/ha would be 0.031 mg/kg (0.024 mg-ha/kg^2 x 1.3 kg/ha). Drift deposition in mg/kg on small fruits or vegetables such as beans, peas, or strawberries is assumed to be 5 percent of the drift deposition on wheatgrass based on a literature review by Hoerger and Kenaga (1972). The difference in concentration is due to the difference in surface to mass ratio. Dosage to humans is then based on assumptions regarding vegetable consumption rates.

The drift at a given distance from a spray site depends on the configuration of the actual treatment area. As discussed in Section 2.1, the spray area is often scattered over an area 10 times greater than the net area sprayed. As a worst-case, it is assumed that the treatment area is continuous and that its nearest boundary is 220 yards (200 meters) from residences. This assumption will overestimate drift concentrations from scattered areas.

Drift data provided by Maybank et al. (1977) are used to determine the impacts of several worst-case exposure scenarios. Because these data lend themselves well to site-specific treatment, they are discussed fully in the section on exposure levels associated with projects involving road rights-of-way and riparian areas (Section 2.4.6).

Drift impacts from a large aerial spray project are based on drift rates calculated for several conditions as provided in a report on the FSCBG model (USDA Forest Service 1984a). This report provides the drift deposition downwind for various combinations of wind speed, air temperature, and spray release height for a 120-acre project sprayed at 10 gallons of herbicide mix per acre containing 0.5 gallons of herbicide. Assuming a wind speed of 10 mph, an air temperature of 90°F, and a spray height of 50 feet, drift deposition at 200 meters would be about 0.16 percent of the application rate on-site (0.0008 gallons per acre/0.5 gallons per acre x 100; see Figure 9 in USDA Forest Service 1984a). Typical specifications for aerial application prohibit application of pesticides when the wind speeds exceed 5 mph. Range aerial application is also typically accomplished at much lower spray release heights which also reduces drift. Thus, these conditions would be expected to produce extremely high drift deposition and will be the basis for "worst-case" drift estimates.

By comparison, drift deposition under more typical conditions (2 mph wind speed, 60°F air temperature, and 10 feet release heights) would be less than 0.1 percent of the drift deposition as calculated above for worst-case conditions (compare Figures 9 and 10 of USDA, Forest Service 1984a). Drift deposition for typical conditions will be based on these later assumptions.

Drift deposition onto vegetation at 200 meters is based on factors developed by Hoerger and Kenaga (1972) with allowances for the differences in on-site application rate and drift deposition off-site. Once again, the drift deposition in mg/kg onto small fruits and vegetables is assumed to be 5 percent of the drift deposition onto leafy vegetation.

Table 2-6. Drift deposition on vegetation at specified distances (from Yates et al. 1978).

Distance (meters)	Drift deposition from a 10-meter-wide spray swath[1]	Drift deposition from a 100-meter-wide spray swath[1]
60	0.100	0.52
100	0.052	0.40
200	0.024	0.21
300	0.017	0.15
400	0.012	0.12
500	0.010	0.10
600	0.008	0.08
700	0.007	0.07
800	0.006	0.06
900	0.006	0.06
1,000	0.005	0.05
1,100	-	0.05[2]
1,200	-	0.04
1,300	-	0.04
1,400	-	0.04
1,500	-	0.03
1,600	-	0.03

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[1] Drift deposition is presented as milligrams of herbicide deposited per kilogram of vegetation when the application rate in kg/ha is multiplied by the appropriate factor from this table. Example: 1.3 kg/ha x .21 mg-ha/kg^2= .273 mg/kg. (Drift deposition at 200 meters from a 100-meter swath sprayed at 1.3 kg/ha.)

[2] Values for 1,100 meters and beyond are extrapolated.

Worst-case dosage to cattle foraging on herbicide-contaminated plants is calculated in these analyses. The dosage figures are compared to controlled feeding studies that measured herbicide intake and retention in cattle and other mammals. Secondary doses to human consumers of herbicide-fed cattle can then be estimated based on assumptions regarding consumption rates.

The dose to a 1,000-pound (450-kilogram) steer consuming 75 lbs/day (35 kgs) of green weight forage that was directly sprayed with herbicide can be estimated as follows. As reported in a literature review by Hoerger and Kenaga (1972), average herbicide concentration on range grass (adjusted to a 1 lb/ac application rate) was 125 mg/kg. Assuming a steer eats forage with an average herbicide concentration of 125 mg/kg, its daily dose would be 9.7 mg/kg (125 mg/kg x 35 kg/steer x steer/450 kg).

To estimate the maximum body burden of herbicide in a steer consuming herbicide-treated forage, the literature on herbicide feeding studies was reviewed. Numerous studies of the herbicides of interest in these risk analyses indicate that, after intake, these herbicides are rapidly excreted from mammalian systems. These studies will be reviewed briefly as a basis for making estimates of herbicide body burdens in cattle.

Khanna and Fang (1966) report 40 to 60 percent elimination of 60 to 100 mg dose of 2,4-D within 24 hours in rats. Cows and sheep fed up to 2,000 ppm 2,4-D in their diet for 28 days had average residue levels of less than 0.6 ppm in muscle, fat, and liver (Clark et al. 1975). At 300 ppm in feed, the 2,4-D residue in muscle was less than 0.05 mg/kg, 0.13 mg/kg in fat, and 0.11 mg/kg in liver. At all concentrations (300, 1,000, and 2,000 ppm), the cattle ate less and refused food occasionally. Feed consumption rates returned immediately to control levels when cattle were withdrawn from dosing.

Dicamba feeding studies in cattle have shown that 60 percent of a dose (60 ppm, dietary) is excreted in 12 hours; that steady state with intake matching excretion is achieved in 2 to 3 days; that the maximum concentration in muscle tissue and fat is 0.03 mg/kg; and that liver concentrations are 0.3 mg/kg or less (Oehler and Ivie 1980).

Picloram is excreted very rapidly from mammalian systems. Nolan et al. (1984) found that more than 70 percent of a human oral dose of 5.0 mg/kg was recovered in urine in 6 hours. Ninety percent of the compound fed to dogs was excreted within 48 hours (Redemann, 1963, reported in National Research Council of Canada 1974 and USDA Forest Service 1984). Cattle fed from 200 to 1,600 ppm of picloram in feed for 4.5 to 8 weeks showed 0.05 to 0.5 mg/kg in muscle and fat, 0.12 to 2.0 mg/kg in liver and 2.0 to 18 mg/kg in kidneys (Kutschinski and Riley 1969). Kidneys contained less than 0.1 mg/kg if picloram was withdrawn from the diet three days before slaughter. At 400 ppm feed levels, muscle concentrations averaged 0.06 mg/kg and perirenal fat averaged 0.09 mg/kg of picloram (Kutschinski and Riley 1969).

Glyphosate is one herbicide which does not follow the typical urinary excretion pathway, primarily because this compound is absorbed only slowly across gastrointestinal membranes. Radiolabeled glyphosate fed in a single dose to rabbits was largely excreted in feces (greater than 80 percent) and to a lesser degree in urine (7 to 11 percent) within 5 days. A small amount (less than 1 percent) was expired as CO_2 or remained in the colon (U.S. Environmental Protection Agency data reported in Ghassemi et al. 1981). Chickens were found to have a bioaccumulation factor as low as 0.0001 for glyphosate in various tissues (Sacher 1978). No storage of radiolabeled glyphosate was found in muscle or fat of bobwhite quail, although traces were found in liver and kidney tissues (USDA Forest Service 1981). Finally, chickens, cows, and pigs fed up to 75 ppm showed nondetectable residues in muscle and fat (less than 0.05 ppm) (Monsanto Company 1982).

Fang et al. (1964, 1966) fed radiolabeled amitrole to rats in concentrations of 1 to 200 mg/rat. From 79 to 89 percent of the total radioactivity administered was found in the urine and feces within 24 hours. Feces contained a small but variable amount of activity. Tissues absorbed material and reached a maximum in 1 hour, but the compound was excreted rapidly with a half-life averaging 4.2 hours in tissue. After a dose of 200 mg (about 500 mg/kg), amitrole levels in muscle tissues and in the stomach were nondetectable within 48 hours. Levels in blood were reduced over 99 percent within 48 hours and levels in liver were reduced almost 90 percent within 48 hours. From these data it can be concluded that very little accumulation in muscle or fat is possible.

Hexazinone fed to goats at 5 ppm in diets showed residues of 0.01 ppm in muscle and fat (Schneider and Kaplan 1983). Milk cows administered 30 daily doses of up to 25 ppm in diet showed no hexazinone in muscle, fat, liver, or kidney at any dose tested (Schneider and Kaplan 1983).

Khan and Foster (1976) have shown no accumulation of atrazine in leg or breast muscle of chickens fed 100 ppm atrazine in diet for 7 days. However, abdominal fat contained 38.8 mg/kg atrazine.

Tebuthiuron would be applied in pellet form and thus would not directly adhere to forage; however, tebuthiuron is absorbed through roots of grasses and is available to grazing animals. In one study conducted in 14 states, tebuthiuron pellets were applied to rangelands at rates varying from 0.5 to 4.0 lb/ac. Maximum concentration in grass was 19.3 ppm (USDA Forest Service 1986a). Maximum tissue levels in cows fed 10 ppm in their diet were 2.0 ppm in the liver and 0.25 in muscle (USDA Forest Service 1986a).

Based on the feeding studies reviewed above it is apparent that very little bioaccumulation of the herbicides of interest occurs in mammalian or avian species, particularly in edible muscle tissue. The only exception is the herbicide atrazine for which there exists some evidence of accumulation in fat cells (though not in muscle tissue). This analysis assumes that the maximum herbicide concentration in cattle feeding on forage treated directly with herbicide is 0.1 mg/kg except for the herbicides atrazine and tebuthiuron. For the herbicide atrazine, 10.0 mg/kg is used in these analyses based on an assumption that edible tissue contain 25 percent fat with a concentration of 40 mg/kg. For the herbicide tebuthiuron, a maximum concentration of 1.0 mg/kg in meat is assumed. It is highly unlikely, of course, that cattle would graze only on herbicide-treated areas, considering the scattered application of herbicides. As discussed in Section 2.3.1, these analyses assume beef that is maximally contaminated with herbicide is the sole source of meat for the residents and any substitution of other meat such as big game would lessen the accumulative dose being considered.

With these general introductory notes, we will now detail the exposure to affected populations from each of the worst-case model projects.

2.4.3 Affected Population, Worst-case Exposure and Dose From Small Projects

2.4.3.1 Worker Dosage from Small Projects

As discussed in Section 2.11, two workers with backpack sprayers could spray a small project in approximately one-quarter day. Under very ideal conditions, they could cover as many as three sites per day with allowances for travel and setup time. Under these conditions, each worker would spray about 1.5 acres (0.6 hectares) per day. In order to calculate the amount of herbicide applied in a day, the typical application rates were used as listed on Table 2-7. Because of the large number of actual field measurements of worker dose, the various mixing and applications errors discussed in Section 2.2 are assumed to be accounted for in the dosage factors provided on Table 2-4.

Table 2-7. Application rates including formulation errors, mixing errors, and swath overlap.

	Typical herbicide application rate		Herbicide applied assuming major mixing error		Herbicide applied assuming minor mixing error	
	Kg/ha	Lb/ac	Kg/ha	Lb/ac	Kg/ha	Lb/ac
2,4-D	2.2	2.0	2.6	2.4	2.4	2.2
Picloram	1.1	1.0	1.3	1.2	1.2	1.1
Dicamba	1.1	1.0	1.3	1.2	1.2	1.1
2,4-D/	0.8	0.75	1.0	0.9	0.9	0.8
Picloram	0.3	0.25	0.3	0.3	0.3	0.27
2,4-D/	0.8	0.75	1.0	0.9	0.9	0.8
Dicamba	0.3	0.25	0.3	0.3	0.3	0.27
Glyphosate	1.1	1.0	1.3	1.2	1.2	1.1
Amitrole	1.1	1.0	1.3	1.2	1.2	1.1
Atrazine	1.1	1.0	1.3	1.2	1.2	1.1
Hexazinone	1.1	1.0	1.3	1.2	1.2	1.1
Tebuthiuron	1.1	1.0	1.3	1.2	1.2	1.1

The daily dosage for a worker spraying small projects with 2,4-D can be calculated by multiplying the daily acreage sprayed (0.6 ha) by the 2,4-D application rate (2.2 kg/ha) by the appropriate dose factor from Table 2-4 (depending on assumptions used). Thus the high worker dosage estimate for the low protection scenario would be 0.23 mg/kg (0.6 ha x 2.2 kg/ha x 1.76 x 10^{-1}). This dosage estimate would be appropriate for a person who applied herbicide with very careless techniques, little regard for personal safety, and wore little protective clothing such as a long sleeve shirt, gloves, and hat.

By contrast, the average 2,4-D dose estimate for a worker applying herbicide using recommended protective techniques would be 0.037 mg/kg (0.6 ha x 2.2 kg/ha x 2.8 x 10^{-2} mg/kg^2). This dose is about one-sixth of the low protection dose (high value).

Table 2-8 summarizes the worker dosage levels for all the herbicides of interest in this analysis. All values of Table 2-8 assume that workers spray three projects in one day (1.5 acres sprayed per worker).

Table 2-8. Worker daily dosage levels from spraying small projects for 1 day (three projects per day).

	Recommended Protection		Low Protection	
	Average Dose 1/	High Dose 1/	Average Dose 1/	High Dose 1/
2,4-D	0.037	0.074	0.114	0.232
Picloram	0.004	0.007	0.011	0.023
Dicamba	0.018	0.037	0.057	0.116
2,4-D/ Picloram	0.013/ 0.001	0.027/ 0.002	0.041/ 0.003	0.084/ 0.006
2,4-D/ Dicamba	0.013/ 0.005	0.027/ 0.010	0.041/ 0.015	0.084/ 0.028
Glyphosate	0.018	0.037	0.057	0.116
Amitrole	0.004	0.007	0.011	0.023
Atrazine	0.018	0.037	0.057	0.116
Tebuthiuron	0.0006	0.0012	--2/	--2/
Hexazinone	0.037	0.074	0.114	0.232

1/ All values are in milligrams/kilograms/day (mg/kg/day).

2/ See discussion in Section 2.4.1

2.4.3.2 General Population Direct Dermal Dose from Drift

The possible dose to the general population must be calculated based on several possible exposure routes. In the case of small, worst-case projects, these routes include dermal absorption of drift deposited on skin, consumption of drift-contaminated vegetables, consumption of beef fed on herbicide-treated forage, dermal absorption from contact with herbicide-treated vegetation, and consumption of herbicide-contaminated wild foods.

In order to calculate drift from a small project, it was assumed that the 1-acre (0.4-hectare) spray zone was a continuous area with dimension of 100 meters by 40 meters. The orientation of this area is assumed to be such that the wind travels along the 100-meter length of the spray zone and the residence is 200 meters directly downwind. From Table 2-5, the drift factor for a 200-meter distance from a 100-meter-wide strip is 0.036 mg-ha/kg-m^2. Drift deposition from a 1.3 kg/ha treatment would be 0.047 mg/m^2 (1.3 kg/ha x 0.036 mg-ha/kg-m^2).

The general population dose values were calculated as follows. Dermal absorption of drift by neighboring residents was calculated on the assumption that all residents were outside their residence during the entire spray period. Adult skin surface area is assumed to be 18.3 ft^2 (1.7 m^2) of which 4 ft^2 (0.37 m^2) is directly exposed (face, neck, "V" of chest, forearms, and hands (based on Davis 1980)).

In order to estimate skin surface area of adolescent and infants, surface area was assumed to be proportional to body weight to the two-thirds power (see Mantel and Schneiderman 1975). Thus,

$$\frac{(W_1)^{2/3}}{(W_2)^{2/3}} = \frac{S_1}{S_2}$$

where W_1 is the weight of an adolescent, W_2 is the weight of an adult, S_1 is the skin surface area of an adolescent, and S_2 is the skin surface area of an adult. Solving for S_1, using the weights assumed in this analysis, gives

$$S_1 = 0.7\ S_2$$

On this basis, an adolescent resident is assumed to have 13 ft^2 (1.2 m^2) of skin of which 3 ft^2 (0.27 m^2) are exposed. In similar fashion, an infant can be estimated to have 4.8 ft^2 (0.45 m^2) of skin, of which 1.6 ft^2 (0.15 m^2) are exposed. All exposed skin is assumed to be directly in the drift pathway and fully exposed to drift (an extreme assumption).

Dermal absorption rate is assumed to be 1 percent for amitrole and picloram; 10 percent for dicamba, glyphosate, tebuthiuron, and 2,4-D; and 20 percent for atrazine and hexazinone. On this basis, the dose to a 70 kg adult from a 1.3 kg/ha application of picloram would be 2.5 x 10^{-6} mg/kg (0.047 mg/m^2 x 0.37

m^2/adult x adult/70 kg x 0.01) or 0.0025 ug/kg (microgram/kilogram). Under similar conditions, dose to children would be 0.0032 ug/kg and dose to infants would be 0.0059 ug/kg.

2.4.3.3 General Population Oral Dosage

Oral dose from consumption of a steer that fed directly on herbicide-treated forage was calculated as follows. The steer was assumed to be slaughtered at the point at which herbicide body burden was at its maximum. As demonstrated in Section 2.4.2, with the possible exceptions of atrazine and tebuthiuron, maximum body burden in edible tissues would not exceed 0.1 mg/kg and very likely would be much less for the herbicides of interest. The dose to human consumers is based on the following assumptions: the steer has a dressed weight of 440 lbs (200 kg); the herbicide concentration in edible parts averages 0.1 mg/kg (1.0 mg/kg for tebuthiuron and 10 mg/kg for atrazine); each adult consumes 1.1 lbs. (0.5 kilograms) of beef per day; each adolescent consumes 0.66 lbs. (0.3 kilograms) of beef per day; each infant consumes 3.5 ounces (0.1 kilograms) of beef per day; the herbicide in beef does not degrade with time or cooking. Based on these assumptions, the daily oral dose of herbicide for a 70 kg adult would be 0.00071 mg/kg (0.5 kg/adult x adult/70 kg x 0.1 mg/kg) or 0.71 ug/kg (microgram/kilogram). The steer would last this family of four about 140 days at these daily consumption rates.

Calculations of oral dose from eating drift-contaminated vegetables are based on measurements by Yates et al. (1978) of drift deposition on wheat plants as discussed in Section 2.4.1. Because wheat plants would typically have a much higher surface-to-mass ratio than garden vegetables, measurements by Yates et al. (1978) will provide a worst-case estimate for leafy vegetables. Once again assuming a 100-meter by 40-meter plot, the drift deposition on vegetables at 200 meters distance would be 0.21 mg/kg for each kilogram of herbicide applied per hectare (from Table 2.6). For a 1.3 kg/ha application, the deposition would be 0.273 mg/kg (1.3 kg/ha x 0.21 mg-ha/kg^2).

As discussed in Section 2.4.2, the concentration on small fruits and vegetables would be approximately 5 percent of the concentration on leafy vegetables. In the example above, the concentration on small fruits and vegetables would be 0.014 mg/kg (0.05 x 0.273 mg/kg).

Based on Total Diet Studies conducted by the Food and Drug Administration (see Gartrell et al. 1985), adults were assumed to consume 17 ounces (0.5 kg) of vegetables from the garden daily. Four ounces (0.10 kg) of these vegetables are assumed to be leafy types and the remainder to be small fruits and vegetables such as beans, peas, or strawberries (0.40 kg). Adolescents were assumed to consume 10 ounces (0.28 kg) of vegetables per day. Of these vegetables, 2 ounces (0.06 kg) are assumed to be leafy vegetables and the remainder, small fruits and vegetables (0.22 kg). Infants are assumed to consume 4 ounces (0.11 kg) of vegetables daily. Of these vegetables, 1 ounce (0.028 kg) is assumed to be leafy vegetables and the remainder, small fruits and vegetables (0.082 kg). Assuming, as a worst-case, that no herbicide was lost in washing or cooking, the dose to a 70-kg adult would be 0.00048 mg/kg ((0.273 mg/kg x 0.10 kg/adult x adult/70 kg) + (0.273 mg/kg x 0.05 x 0.40 kg x adult/70 kg)). This dose is equivalent to 0.48 ug/kg (microgram/kilogram). Adolescent and infant doses would be 0.00048 mg/kg and 0.00067 mg/kg, respectively (0.48 ug/kg and 0.67 ug/kg).

2.4.3.4 Re-entry Doses

Several studies of herbicide residue in treated areas indicate that the herbicide exposure to persons entering a spray area, after spraying has been completed, will be very small. Lavy et al. (1980a) reported that individuals who walked through an area sprayed 2 hours earlier with 2,4,5-T had no detectable dislodgable residue levels on patches which represented dermal exposure to skin and clothing. Also, Thompson et al. (1983) found that only 5 percent of 2,4-D applied to grasses could be removed by mechanical wiping immediately after spraying 1 to 2 lb a.e/acre. These residues dropped to less than 1 percent by five days after application. These data indicate that the exposure to herbicides from contacting treated foliage would be extremely small.

As an estimate of dose to a visitor of a model spray site, the high dose levels measured for a spray project supervisor who had spent a day on-site during spray application will be used. As shown on Table 2-3, the supervisor high dose factor for amitrole and picloram is 2.2×10^{-5} mg/kg per kilogram of herbicide applied; for 2,4-D, dicamba, atrazine, and glyphosate is 1.1×10^{-4} mg/kg; and for hexazinone is 2.2×10^{-4} mg/kg. Because tebuthiuron is applied only as pellets and thus would not be contacted dermally on vegetation, the tebuthiuron dose factor is assumed to be 1 percent of the 2,4-D factor (see also Section 2.4.1). Visitor dose from reentry to a small project sprayed with picloram at 1.3 kg/ha would be 0.000011 mg/kg (1.3 kg/ha x 0.4 ha x 2.2×10^{-5} mg/kg/ha) or 0.011 ug/kg (microgram/kilogram).

The dose to a Forest visitor who collects and eats 0.5 pound (0.23 kg) of wild fruit from a spray site was calculated on the basis of studies reported by Norris (1981) and Hoerger and Kenaga (1972). They reported the upper concentration limits on fruit would be about 7 mg/kg for each pound of herbicide applied per acre, or about 6.5 mg/kg for each kilogram of herbicide applied per hectare. The dose to a 70 kg person eating wild fruit from a site sprayed at 1.3 kg/ha would be 0.028 mg/kg (1.3 kg/ha x 6.5 mg-ha/kg^2 x 0.23 kg/70 kg) or 28 ug/kg. The chances of picking and consuming one-half pound of wild foods exclusively from treatment areas that have been directly sprayed with herbicides are extremely small. It is even less likely that a person would pick and consume wild foods from an area that has been mistakenly sprayed with a swath overlap or over-strength batch of herbicide mixture.

Tables 2-9 through 2-15 provide general population dose estimates from the spraying of each herbicide or mixture of herbicides on small projects.

Text continued on page 56.

Table 2-9. Daily dosage to visitors and residents in the vicinity of small projects sprayed with 2,4-D.[1/]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.046	0.050
Adolescent dermal dose	0.058	0.063
Infant dermal dose	0.108	0.117
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.88	0.96
Infant oral dose (veg)	1.24	1.34
Visitor entry to spray site	0.11	0.12
Oral dose/sprayed wild food	51	56

[1/] Based on a prescribed application rate of 2.0 lbs/ac. Minor mixing error scenario assumes an application rate of 2.2 lbs/ac (2.4 kg/ha). Major mixing error assumes an application rate of 2.4 lbs/ac (2.6 kg/ha). Dermal dose assumes an absorption rate of 10 percent. Additional assumptions:

	Body wt. kg	Exposed skin m^2	Daily veg. consumption kg	Daily beef consumption kg
adults	70 kg	0.37	0.5	0.5
adoles.	40 kg	0.27	0.28	0.3
infant	12 kg	0.15	0.11	0.1

Table 2-10. Daily dosage to visitors and residents in the vicinity of small projects sprayed with picloram or amitrole.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.002	0.003
Adolescent dermal dose	0.003	0.003
Infant dermal dose	0.005	0.006
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.44	0.48
Infant oral dose (veg)	0.62	0.67
Visitor entry to spray site	0.011	0.012
Oral dose/sprayed wild food	26	28

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[1] Based on a prescribed application rate of 1.0 lb/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Dermal dose assumes an absorption rate of 1 percent.

Table 2-11. Daily dosage to visitors and residents in the vicinity of small projects treated with hexazinone.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.046	0.049
Adolescent dermal dose	0.058	0.063
Infant dermal dose	0.108	0.117
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.44	0.48
Infant oral dose (veg)	0.62	0.67
Visitor entry to spray site	0.11	0.12
Oral dose/sprayed wild food	26	28

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[1] Based on a prescribed application rate of 1.0 lb/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Dermal dose assumes an absorption rate of 20 percent.

Table 2-12. Daily dosage to visitors and residents in the vicinity of small projects sprayed with dicamba or glyphosate.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.023	0.025
Adolescent dermal dose	0.029	0.032
Infant dermal dose	0.054	0.059
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.44	0.48
Infant oral dose (veg)	0.62	0.67
Visitor entry to spray site	0.053	0.057
Oral dose/sprayed wild food	26	28

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[1] Based on a prescribed application rate of 1.0 lb/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Dermal dose assumes an absorption rate of 10 percent.

Table 2-13. Daily dosage to visitors and residents in the vicinity of small projects sprayed with a 2,4-D/picloram mixture.[1/]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.017	0.019
	0.001	0.001
Adolescent dermal dose	0.022	0.024
	0.001	0.001
Infant dermal dose	0.041	0.044
	0.001	0.002
Adult/adolescent oral dose (beef)	0.71	0.71
	0.71	0.71
Infant oral dose (beef)	0.83	0.83
	0.83	0.83
Adult/adolescent oral dose (veg)	0.33	0.35
	0.11	0.12
Infant oral dose (veg)	0.46	0.50
	0.16	0.17
Visitor entry to spray site	0.040	0.042
	0.003	0.003
Oral dose/sprayed wild food	19	21
	6.4	7.0

[1/] This table is based on a prescribed application rate of 1.0 lb/ac (1.1 kg/ha) of the herbicide mixture with allowances for mixing errors. The mixture is comprised of 2,4-D and picloram in a 3 to 1 ratio.

Table 2-14. Daily dosage to visitors and residents in the vicinity of small projects sprayed with a 2,4-D/dicamba mixture.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.017 0.006	0.019 0.006
Adolescent dermal dose	0.022 0.007	0.024 0.008
Infant dermal dose	0.042 0.014	0.044 0.015
Adult/adolescent oral dose (beef)	0.71 0.71	0.71 0.71
Infant oral dose (beef)	0.83 0.83	0.83 0.83
Adult/adolescent oral dose (veg)	0.33 0.11	0.35 0.12
Infant oral dose (veg)	0.46 0.16	0.50 0.17
Visitor entry to spray site	0.040 0.013	0.042 0.014
Oral dose/sprayed wild food	19 6.4	21 7.0

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[1] This table is based on an application rate of 1.0 lb/ac (1.1 kg/ha) of the herbicide mixture with allowances for mixing errors. Mixture is comprised of 2,4-D and dicamba in a 3 to 1 ratio.

Table 2-15. Daily dosage to visitors and residents in the vicinity of small projects sprayed with atrazine or treated with tebuthiuron.[1]

	Atrazine dosage including minor mixing error (microgram/ kilogram/day)	Atrazine dosage including major mixing error (microgram/ kilogram/day)	Tebuthiuron dosage including major mixing error (microgram/ kilogram/day)
Adult dermal dose	0.046	0.050	NA[2]
Adolescent dermal dose	0.058	0.063	NA[2]
Infant dermal dose	0.108	0.117	NA[2]
Adult/adolescent oral dose (beef)	71	71	7.1
Infant oral dose (beef)	83	83	8.3
Adult/adolescent oral dose (veg)	0.44	0.48	NA[2]
Infant oral dose (veg)	0.62	0.67	NA[2]
Visitor entry to spray site	0.053	0.057	0.00057
Oral dose/sprayed wild food	26	28	NA[3]

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[1] Based on a prescribed application rate of 1.0 lb/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Atrazine dermal dose assumes an absorption rate of 20 percent.

[2] Tebuthiuron will be applied in pellet form. Thus, the surrounding areas would not be exposed to drift.

[3] Uptake of tebuthiuron in the roots of fruit-bearing shrubs and plants and other wild foods would prevent development.

2.4.4 Affected Population Exposure and Dose from Mid-Sized Projects

The doses to workers and the general population from mid-sized projects are calculated using the same basic methods discussed for small projects.

2.4.4.1 Worker Dosage from Mid-Sized Projects

As discussed in Section 2.3.2, it is assumed that four workers with backpack sprayers spend 6 days each on this project. Thus, each applicator would treat approximately 1.67 net acres (0.68 net hectares) per day. A range of worker doses was again calculated by multiplying the kilograms of herbicide applied per day by the dose factors provided on Table 2-4. For example, the high dose to workers using recommended protective techniques while applying picloram at 1.1 kg/ha would be 0.008 mg/kg (0.68 ha x 1.1 kg/ha x 1.1 x 10^{-2} mg/kg/kg). Table 2-16 provides these daily dosages for workers based on the typical application rates shown in Table 2-7.

Table 2-16. Worker daily dosage levels from spraying mid-sized projects.

	Recommended Protection		Low Protection	
	Average Dose[1]	High Dose[1]	Average Dose[1]	High Dose[1]
2,4-D	0.042	0.084	0.129	0.263
Picloram	0.005	0.008	0.012	0.026
Dicamba	0.020	0.042	0.065	0.131
2,4-D/ Picloram	0.015/ 0.001	0.031/ 0.002	0.046/ 0.003	0.095/ 0.007
2,4-D/ Dicamba	0.015/ 0.006	0.031/ 0.011	0.046/ 0.017	0.095/ 0.036
Glyphosate	0.020	0.042	0.065	0.131
Amitrole	0.005	0.008	0.012	0.026
Atrazine	0.020	0.042	0.065	0.131
Hexazinone	0.042	0.084	0.129	0.263
Tebuthiuron	0.0007	0.0013	--[2]	--[2]

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[1] All values are in milligram/kilogram/day (mg/kg/day).

[2] See discussion in Section 2.4.1.

2.4.4.2 General Population Dosage from Drift on Mid-sized Projects

Spraying will be treated as though it occurs daily on a continuous 2.7-hectare site (6.6-acres) with dimensions of 200 meters by 135 meters. The spray zone is assumed to be oriented such that the wind blows directly along the 200 meter length of the spray zone. Drift is calculated on the basis that half of the project is 200 meters (220 yards) from the residence and half is 300 meters from the house (using factors from Table 2-5). Drift deposition at 200 meters from a day's spraying at 1.3 kg/ha (1.2 lbs/ac) would be 0.078 mg/m^2 (1.3 kg/ha x (0.036 $mg\text{-}ha/kg\text{-}m^2$ + 0.024 $mg\text{-}ha/kg\text{-}m^2$)).

Dermal absorption rates and assumptions regarding area of exposed skin are identical to those used for small projects. Daily doses from spray areas further than 200 to 300 meters would be proportionately smaller.

2.4.4.3 General Population Oral Doses

The oral doses from consumption of beef fed on herbicide-treated forage are the same as for small projects in section 2.4.3. In both cases, it is assumed that the beef were fed on herbicide-treated forage to the point of maximum herbicide body-burden, i.e. steady state, where herbicide intake is matched by excretion. In reality, because of the scattered nature of herbicide applications and the tendency of cattle to graze at random, the actual dose to beef cattle and subsequent dose to humans would be much less than that indicated for either small or mid-sized projects.

As with the dermal dose to residents near mid-sized projects, the oral dose to residents from drift-contaminated vegetables is calculated on the basis that a day's spraying covers a continuous area of dimensions 220 yards x 150 yards (200 meters x 135 meters). Drift deposition factors of 0.21 and 0.15 $mg\text{-}ha/kg^2$ from Table 2-6 for the distances of 200 meters and 300 meters are combined for a factor of 0.36 $mg\text{-}ha/kg^2$ when calculating concentration on vegetation. Drift deposition on vegetation downwind from a site sprayed at 1.3 kg/ha would be 0.47 mg/kg of vegetation (0.36 $mg\text{-}ha/kg^2$ x 1.3 kg/ha). Daily dose to a 70-kg adult consuming 17 ounces (0.5 kg) of vegetables (consisting of 4 ounces of leafy vegetables and 13 ounces of small fruits and vegetables) would be 0.00081 mg/kg ((0.47 mg/kg x 0.10 kg/adult x adult/70 kg) + (0.47 mg/kg x 0.05 x 0.40 kg/adult x adult/70 kg)). This dose is equivalent to 0.81 ug/kg (microgram/kilogram).

2.4.4.4 Re-entry Doses

Visitor doses are calculated as for small projects in Section 2.4.3. Because the mid-sized project would afford more opportunity for exposure in a day than the smaller projects, the dosage estimates for visitor reentry were increased proportionately. For example, the dosage estimate for a visitor reentering a daily spray area on a mid-sized project sprayed with picloram at 1.3 kg/ha would be 7.7 x 10^{-5} mg/kg (2.7 ha x 1.3 kg/ha x 2.2 x 10^{-5} mg/kg/kg) or 0.077 ug/kg.

The dose to a visitor who consumes sprayed wild food is also calculated as with small projects. As shown in Section 2.4.3, oral doses of 0.028 mg/kg of body weight could be expected from the consumption of 0.5 pounds (0.23 kg) of wild foods collected from an area treated at a rate of 1.3 kg of herbicide per hectare.

As discussed in Section 2.4.3, visitor dosages estimates are extreme. The chances of picking and consuming one-half pound of wild foods exclusively from treatment areas that have been directly sprayed with herbicides are extremely small. Even smaller is the probability of a person picking and consuming wild foods from an area that has been mistakenly sprayed with an over-strength batch of herbicide mixture. The odor and taste of the sprayed vegetation alone should alert the person to contamination. (Text continued on page 65)

Table 2-17. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with 2,4-D.[1/]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.08	0.08
Adolescent dermal dose	0.10	0.11
Infant dermal dose	0.18	0.20
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	1.53	1.62
Infant oral dose (veg)	2.12	2.30
Visitor entry to spray site	0.71	0.78
Oral dose/sprayed wild food	51	56

[1/] Based on a prescribed application rate of 2 lbs/ac. Minor mixing error scenario assumes an application rate of 2.2 lbs/ac (2.4 kg/ha). Major mixing error assumes an application rate of 2.4 lbs/ac (2.6 kg/ha). Dermal dose assumes an absorption rate of 10 percent.

Table 2-18. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with picloram or amitrole.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.004	0.004
Adolescent dermal dose	0.005	0.005
Infant dermal dose	0.009	0.010
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.77	0.81
Infant oral dose (veg)	1.06	1.15
Visitor entry to spray site	0.07	0.08
Oral dose/sprayed wild food	26	28

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[1] Based on a prescribed application rate of 1.0 lbs/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Dermal dose assumes an absorption rate of 1 percent.

Table 2-19. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with hexazinone.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.08	0.08
Adolescent dermal dose	0.10	0.11
Infant dermal dose	0.18	0.20
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.77	0.83
Infant oral dose (veg)	1.06	1.15
Visitor entry to spray site	0.71	0.78
Oral dose/sprayed wild food	26	28

---

[1] Based on a prescribed application rate of 1.0 lbs/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Dermal dose assumes an absorption rate of 20 percent.

Table 2-20. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with dicamba or glyphosate.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.04	0.04
Adolescent dermal dose	0.05	0.05
Infant dermal dose	0.09	0.10
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	0.77	0.83
Infant oral dose (veg)	1.06	1.15
Visitor entry to spray site	0.35	0.39
Oral dose/sprayed wild food	26	28

---

[1] Based on a prescribed application rate of 1.0 lbs/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Dermal dose assumes an absorption rate of 10 percent.

Table 2-21. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with a 2,4-D/picloram mixture.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.03 0.001	0.03 0.001
Adolescent dermal dose	0.04 0.001	0.04 0.001
Infant dermal dose	0.07 0.002	0.07 0.002
Adult/adolescent oral dose (beef)	0.71 0.71	0.71 0.71
Infant oral dose (beef)	0.83 0.83	0.83 0.83
Adult/adolescent oral dose (veg)	0.58 0.19	0.62 0.21
Infant oral dose (veg)	0.80 0.27	0.86 0.29
Visitor entry to spray site	0.27 0.02	0.28 0.02
Oral dose/sprayed wild food	19 6.4	21 7.0

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[1] This table is based on an application rate of 1.0 lb/ac (1.1 kg/ha) of the herbicide mixture with allowances for mixing errors. Mixture is comprised of 2,4-D and picloram in a 3 to 1 ratio.

Table 2-22. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with a 2,4-D/dicamba mixture.[1]

	Dosage including minor mixing error (microgram/kilogram per day)	Dosage including major mixing error (microgram/kilogram per day)
Adult dermal dose	0.03 0.01	0.03 0.01
Adolescent dermal dose	0.04 0.01	0.04 0.01
Infant dermal dose	0.07 0.02	0.07 0.03
Adult/adolescent oral dose (beef)	0.71 0.71	0.71 0.71
Infant oral dose (beef)	0.83 0.83	0.83 0.83
Adult/adolescent oral dose (veg)	0.58 0.19	0.62 0.21
Infant oral dose (veg)	0.80 0.27	0.86 0.29
Visitor entry to spray site	0.27 0.09	0.28 0.09
Oral dose/sprayed wild food	19 6.4	21 7.0

---

[1] This table is based on an application rate of 1.0 lb/ac (1.1 kg/ha) of the herbicide mixture with allowances for mixing errors. Mixture is comprised of 2,4-D and dicamba in a 3 to 1 ratio.

Table 2-23. Daily dosage to visitors and residents in the vicinity of mid-sized projects sprayed with atrazine or treated with tebuthiuron.[1]

	Atrazine dosage including minor mixing error (microgram/ kilogram/day)	Atrazine dosage including major mixing error (microgram/ kilogram/day)	Tebuthiuron dosage including major mixing error (microgram/ kilogram/day)
Adult dermal dose	0.08	0.08	NA[2]
Adolescent dermal dose	0.10	0.11	NA[2]
Infant dermal dose	0.18	0.20	NA[2]
Adult/adolescent oral dose (beef)	71	71	0.71
Infant oral dose (beef)	83	83	0.83
Adult/adolescent oral dose (veg)	0.77	0.81	NA[2]
Infant oral dose (veg)	1.06	1.15	NA[2]
Visitor entry to spray site	0.35	0.39	0.0039
Oral dose/sprayed wild food	26	28	NA[3]

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[1] Based on a prescribed application rate of 1.0 lb/ac. Minor mixing error scenario assumes an application rate of 1.1 lbs/ac (1.2 kg/ha). Major mixing error assumes an application rate of 1.2 lbs/ac (1.3 kg/ha). Atrazine dermal dose assumes an absorption rate of 20 percent.

[2] Tebuthiuron will be applied in pellet form. Thus, the surrounding areas would not be exposed to drift.

[3] Uptake of tebuthiuron in the roots of fruit-bearing shrubs and plants and other wild foods would prevent development.

2.4.5 Affected Population, Exposure and Dose from Large Ground Application Projects

Large projects will most often be treated with vehicle-mounted spray equipment. Areas inaccessible to vehicles may be treated with backpack sprayers.

As a basis for calculating worst-case worker doses from large projects, it is assumed that 20 percent (100 acres) of the large project is treated by seven workers (six applicators and one supervisor) for 10 work days each. This application rate is equivalent to 1.67 net acres (0.67 hectares) per applicator per day. The remainder of the project is treated in 10 days with a vehicle-mounted spray rig by one driver who also does his own mixing and loading of herbicide.

Table 2-7 provides application rates for herbicide mixes most likely used on large-scale projects. Glyphosate, amitrole, atrazine, and hexazinone are not likely to be used on large projects.

As discussed in Section 2.2, major mixing error overstrength applications are assumed to occur on the acreage treated with backpack sprayers. Major mixing error is not considered with vehicle application because of the large and obvious increase in herbicide consumption which would result. A 4 percent excess formulation error, a 10 percent field mixing error, and a 5 percent swath overlap is assumed for all vehicle applications (i.e., the minor mixing error scenario).

Tables 2-24 and 2-25 present the estimates for worker daily doses for large projects. As with small and mid-sized projects, backpack worker dose was calculated by adding the area treated per day (0.67 net hectares or 1.67 net acres) by the prescribed herbicide application rate per acre (see Table 2-7) and by the backpack dose factors from Table 2-4. The truck driver dose was calculated by multiplying the area treated per day, (16 hectares or 40 acres) by the prescribed application rate from Table 2-7 and by the appropriate truck driver/mixer/loader factors from Table 2-3. Since the supervisor would be affected, both by vehicle application and backpack spray, his daily dose was calculated on a 20-hectare or 50-acre daily spray basis. As can be seen on Table 2-25, the supervisor dose is much smaller than either the backpack worker or truck driver dose. On this basis it can be safely assumed that the effect of the backpack spray drift on the truck driver (or the reverse) would be negligible in comparison to the dose from his own specific occupational activities.

A range of four doses is provided for backpack spray applicators; whereas, only average and high doses are given for truck drivers and supervisors. As discussed in Section 2.4.1, the data base for truck driver and supervisor exposure levels is representative of the conditions under which most Forest Service workers treat target plants. Because the backpack/spot sprayer data for workers was based on extreme working conditions and because personal worker habits can effect worker dose to a great degree (as seen in the large range of doses in Lavy et al. 1984), a greater range of dose estimates is provided for spot sprayers.

2.4.5.1 General Population Dosage from Drift on Large Projects (Ground Application)

Tables 2-26 through 2-28 present dose levels for residents and visitors in the vicinity of these large projects. As discussed in Section 2.1.3, a residence is assumed at 220 yards (200 meters) from the nearest edge of each of the large projects. Further, it is assumed that the 500-acre (200-hectare) project is treated in ten strips of 140-meters width by 1,400 meters (one strip sprayed each day). Finally, it is assumed that the wind blows parallel with the long axis of the treated area and that the residence is directly downwind of a daily treatment area.

This long and narrow pattern oriented with the long axis into the wind will provide the highest daily drift deposition from this large project. The orientation is in keeping with the tenor of a worst-case scenario.

Drift from each of the daily treatment areas (1,400 by 140 meters) is calculated using data from Table 2-5 for 100-meter-wide strips. Total daily drift from a large project can be calculated by adding the drift factors from Table 2-5 for 200 through 1,600 yards and then multiplying this sum by the application rate. Application rate is assumed to be the prorated average of 4 hectares sprayed with backpack sprayers with major mixing errors and 16 hectares sprayed with trucks with minor errors. Thus, the assumed application rate from a prescribed rate of 1.1 kg/ha would be 1.22 kg/ha ((0.8 x 1.2 kg/ha) + (0.2 x 1.3 kg/ha)).

Based on these assumptions, total daily drift deposition on surfaces at 220 yards (200 meters) distance from a large project treated at 1.22 kg/ha would be 0.17 mg/m^2 ((0.036 + 0.024 + 0.017 + . . . + 0.003 + 0.002 + 0.002) mg-ha/kg-m^2 x 1.22 kg/ha)). Dermal absorption is calculated assuming that the residents are outside the entire day during treatment, that the adults have 4 ft^2 (0.37 m^2) of exposed skin, adolescents, 3 ft^2 (0.27 m^2), and infants, 1.6 ft^2 (0.15 m^2). The dermal absorption rate for picloram and amitrole is assumed to be 1 percent; for atrazine and hexazinone, 20 percent; and all others, 10 percent. Tables 2-26 through 2-28 provide worst-case dermal dose levels for adults, adolescents, and infants.

2.4.5.2 General Population Oral Dosages from Large Projects (Ground Application)

Worst-case oral doses to humans from eating cattle that have grazed on herbicide-treated forage are calculated in an identical fashion as for small and mid-sized projects. These dose values are also provided on Tables 2-26 through 2-28.

The worst-case dose from eating drift-contaminated vegetables from a garden is calculated in a similar fashion to the dermal doses from drift. Drift deposition on vegetables is calculated by combining factors for 100-meter-wide swaths from Table 2-6 for 200 through 1,600 meter distances. Daily vegetable consumption rates were the same as small and mid-sized projects. Once again, no loss of herbicide was assumed during washing and cooking.

2.4.5.3 General Population, Visitor Oral Dosage from Large Projects (Ground Application)

Tables 2-26 through 2-28 also provide worst-case dose levels for visitor entry to treatment sites and visitor consumption of wild food gathered from these sites. Dose from visitor reentry is assumed to be the same as the supervisor dose as discussed in Section 2.4.3.4. The dose from wild food consumption is unlikely because, aside from the fact that a small percentage of NFS land will be sprayed, those sites have little or no vegetation that is edible by humans, e.g., huckleberries. The odor and taste of edible wild food treated at concentrations necessary to give the doses reported here may also be objectionable and diminish the possibility of human consumption.

Table 2-24. Daily dosage for backpack workers on large projects.

	Recommended Protection		Low Protection	
	Average Dose 1/	High Dose 1/	Average Dose 1/	High Dose 1/
2,4-D	0.042	0.084	0.129	0.263
Picloram	0.005	0.008	0.012	0.026
Dicamba	0.020	0.042	0.065	0.131
2,4-D/ Picloram	0.015/ 0.001	0.031/ 0.002	0.046/ 0.003	0.095/ 0.007
2,4-D/ Dicamba	0.015/ 0.006	0.031/ 0.011	0.046/ 0.017	0.095/ 0.036
Glyphosate	0.020	0.042	0.065	0.131
Amitrole	0.005	0.008	0.012	0.026
Atrazine	0.020	0.042	0.065	0.131
Hexazinone	0.042	0.084	0.129	0.263
Tebuthiuron	0.0007	0.0013	--2/	--2/

1/ All values are in milligram/kilogram/day (mg/kg/day).

2/ See discussion in Section 2.4.1.

Table 2-25. Daily dosage for truck drivers and supervisors on large projects.

	Supervisor dose[1]		Truck driver dose	
	Average	High	Average	High
2,4-D	0.0011	0.0048	0.030	0.113
2,4-D/ Picloram	0.0004/ 0.00003	0.0018/ 0.0001	0.011/ 0.0007	0.042/ 0.003
2,4-D/ Dicamba	0.0004/ 0.0001	0.0018/ 0.0006	0.011/ 0.004	0.042/ 0.013
Glyphosate	0.0005	0.0024	0.015	0.056
Amitrole	0.0001	0.0005	0.003	0.011
Atrazine	0.0005	0.0024	0.015	0.056
Dicamba	0.0005	0.0024	0.015	0.056
Hexazinone	0.0011	0.0048	0.030	0.113
Picloram	0.0001	0.0005	0.003	0.011
Tebuthiuron	0.000005[2/]	0.000024	0.0002	0.0006

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[1] All dose values are in milligram/kilogram/day (mg/kg/day).

[2/] Tebuthiuron dose factor is assumed to be 1 percent of 2,4-D factor (with allowances for application rate). See Section 2.4.1.

Table 2-26. Daily dosage to visitors and residents in the vicinity of large projects treated with 2,4-D, picloram, amitrole, or dicamba.[1/]

	2,4-D (microgram/ kilogram/day)	Amitrole or Picloram (microgram/ kilogram/day)	Dicamba (microgram/ kilogram/day)
Adult dermal dose	0.18	0.01	0.09
Adolescent dermal dose	0.23	0.01	0.12
Infant dermal dose	0.43	0.02	0.21
Adult/adolescent oral dose (beef)	0.71	0.71	0.71
Infant oral dose (beef)	0.83	0.83	0.83
Adult/adolescent oral dose (veg)	4.7	2.4	2.4
Infant oral dose (veg)	7.4	3.7	3.7
Visitor entry to spray site	1.1	0.1	0.5
Oral dose/sprayed wild food	51	26	26

[1/] Application rates in lbs/ac or kg/ha are identical to small and mid-sized open range projects.

Table 2-27. Daily dosage to visitors and residents in the vicinity of large projects treated with a 2,4-D/picloram or 2,4-D/dicamba mixture.[1/]

	2,4-D (microgram/ kilogram/day)	Picloram (microgram/ kilogram/day)	Dicamba (microgram/ kilogram/day)
Adult dermal dose	0.07	0.002	0.02
Adolescent dermal dose	0.09	0.003	0.03
Infant dermal dose	0.17	0.005	0.05
Adult/adolescent oral dose (beef)	0.71	0.71	0.71
Infant oral dose (beef)	0.83	0.83	0.83
Adult/adolescent oral dose (veg)	1.8	0.6	0.6
Infant oral dose (veg)	2.8	0.9	0.9
Visitor entry to spray site	0.4	0.03	0.05
Oral dose/sprayed wild food	20	6.0	6.0

[1/] Application rates in lbs/ac or kg/ha are identical to small and mid-sized open range projects.

Table 2-28. Daily dosage to visitors and residents in the vicinity of large projects treated with glyphosate, hexazinone, tebuthiuron, or atrazine.[1]

	Glyphosate (microgram/ kilogram/day)	Hexazinone (microgram/ kilogram/day)	Atrazine (microgram/ kilogram/day)	Tebuthiuron (microgram/ kilogram/day)
Adult dermal dose	0.09	0.18	0.18	NA[2]
Adolescent dermal dose	0.12	0.23	0.23	NA[2]
Infant dermal dose	0.21	0.43	0.43	NA[2]
Adult/adolescent oral dose (beef)	0.71	0.71	71	7.1
Infant oral dose (beef)	0.83	0.83	83	8.3
Adult/adolescent oral dose (veg)	2.4	2.4	2.4	NA[2]
Infant oral dose (veg)	3.7	3.7	3.7	NA[2]
Visitor entry to spray site	0.5	1.1	0.5	0.0039
Oral dose/ sprayed wild food	26	26	26	NA[3]

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[1] Application rates in lbs/ac or kg/ha are identical to small and mid-sized open range projects.

[2] Tebuthiuron will be applied in pellet form. Thus the surrounding area would not be exposed to drift.

[3] The uptake of tebuthiuron in the roots of fruit-bearing shrubs and plants and other wild foods would prevent development.

2.4.6 Affected Population Exposure and Dose from Right-of-Way/Riparian Projects

2.4.6.1 Worker Doses

As discussed in Section 2.1.4, the model road right-of-way treatment project is expected to require one truck driver and a co-worker who spot treats the travelway areas with a hand-held spray nozzle or backpack. Each project is assumed to involve 10 miles of roadside treated on a 20-foot (6-meter) swath (both sides of 5 miles or 8 kilometers of road). The truck is assumed to treat 9 hectares (22 acres) and the spot sprayer treats 0.8 hectares (2 acres).

Daily dosage for the two workers are included in Tables 2-29 and 2-30. As with other projects, the worker dose was calculated by multiplying the prescribed daily application amount (Table 2-7) by the dose factors for a truck driver or a backpack worker from Tables 2-3 and 2-4. The truck driver was assumed to be exposed to the entire amount of herbicide applied to each 24-acre project. For example, the truck driver's average dose for a 1.1 kg/ha application of dicamba would be 0.009 mg/kg (9.8 ha x 1.1 kg/ha x 0.00085 mg/kg/kg). The average spot sprayer dose, assuming recommended protection gear, would be 0.025 mg/kg (0.8 ha x 1.1 kg/ha x 0.028 mg/kg/kg).

Table 2-29. Dosage levels for truck drivers spraying right-of-way projects for 1 day.

	Truck driver dose[1]	
	Average	High
2,4-D	0.02	0.08
Picloram	0.002	0.008
Dicamba	0.01	0.04
2,4-D/Picloram	0.008/0.0005	0.03/0.002
2,4-D/Dicamba	0.008/0.003	0.03/0.01
Glyphosate	0.01	0.04
Amitrole	0.002	0.008
Atrazine	0.01	0.04
Hexazinone	0.02	0.08
Tebuthiuron	0.0001	0.0004

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[1] All doses are in milligrams/kilogram/day (mg/kg/day)

Table 2-30. Daily dosage for backpack workers on right-of-way projects.[1]

	Recommended Protection		Low Protection	
	Average Dose[1]	High Dose[1]	Average Dose[1]	High Dose[1]
2,4-D	0.049	0.099	0.152	0.309
Picloram	0.005	0.010	0.015	0.031
Dicamba	0.025	0.049	0.076	0.154
2,4-D/ Picloram	0.017/ 0.001	0.037/ 0.002	0.057/ 0.004	0.112/ 0.007
2,4-D/ Dicamba	0.017/ 0.007	0.037/ 0.012	0.057/ 0.019	0.112/ 0.037
Glyphosate	0.025	0.049	0.076	0.154
Amitrole	0.005	0.010	0.015	0.031
Atrazine	0.025	0.049	0.076	0.154
Tebuthiuron	0.0008	0.0015	NA[2]	NA[2]
Hexazinone	0.0049	0.099	0.152	0.309

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[1] All values are in milligram/kilogram/day (mg/kg/day).

[2] See discussion in Section 2.4.1.

A range of four doses is provided for spot sprayers, whereas only average and high doses are given for truck drivers. As discussed in Section 2.4.1, the data base for truck drivers and supervisor exposure levels is representative of the conditions under which most Forest Service workers treat noxious weeds. Because the backpack/spot sprayer data for workers was based on extreme working conditions and because personal worker habits can effect worker dose to a great degree (as seen in the large range of doses in Lavy et al. 1984), a greater range of dose estimates is provided for spot sprayers.

In addition, the use of dose factors for backpack workers will give a high estimate of the dose to the worker using the hand-held nozzle on travelway projects. This worker would not be involved in the frequent mixing of spray solutions because he would fill the backpack from the larger spray tank or he would be spraying with a spray nozzle and hose connected directly to the larger spray tank.

2.4.6.2 General Population Dermal Dose from Drift

The dose to residents was calculated on the assumption, discussed in Section 2.3.4, that a residence with four inhabitants is located about 225 feet or 60 meters downwind of the treatment project. As with the other model treatment areas, all residents are assumed to be outdoors during the entire project. In addition, the adolescent is assumed to be attracted by the sound and sight of the treatment equipment and to approach and stand immediately adjacent to the travelway during treatment.

Dermal dose values for residents who are 200 feet (60 meters) from a treated area are calculated as were dermal doses from drift from other projects. Drift deposition factors for 200 feet (60 meters) are available on Table 2-5.

The drift deposition factor from Table 2-5 is adjusted for the fact that the travelway treatment areas involve two 6-meters wide swaths (see 2.3.4) and the drift factors in Table 2-5 are based on a 10-meter wide swath. The drift deposition factor is adjusted by a multiplier of 1.2 (12 meters/10 meters).

Assuming 0.37 m^2 of exposed skin, a 10 percent dermal absorption rate, and an application rate of 1.2 kg/ha rate, the dermal dose to a 70 kg person would be 1.9 x 10^{-5} mg/kg (1.2 kg/ha x (1.2 x 0.025 mg-ha/kg-m^2) x 0.10 x 0.37 m^2 x adult/70 kg) or 0.019 ug/kg (microgram/kilogram).

There are several ways to estimate the dose to an adolescent in the immediate vicinity of a treatment area. One method is to assume that this exposure would not be greater than a supervisor or project observer who spends an entire day on a treatment area. Since, by comparison, a bystander's exposure would be transient, his dose should be less than that of a supervisor for large projects.

A second method of estimating dose to a bystander will provide an even higher estimate. Maybank et al. (1977) made numerous tests to measure deposition on target as well as deposition within 16 feet (5 meters) off-target during application by ground rigs. In the 30 trials with wind speeds up to 20 mph, the highest concentration drift cloud measured with air samplers at 3 feet (1 meter) from the treatment site was equivalent to 25.2 mg/m^2. (Note that Maybank et al. express air sampler data as mass per unit area, i.e., deposition on the sampler filter. This sampling method tends to overestimate deposition onto surfaces such as vegetation or human skin.). This drift deposition resulted from an application of 0.5 lb/ac (.56 kg/ha) on a 45-foot (13.7-meters) wide swath. The highest level of herbicide deposited on an area 3 feet from a 45-foot wide spray swath is about 45 percent of the nominal on-site application rate (56 mg/m^2).

Since the swath width of concern in a travelway project is 20 feet as opposed to 45 feet, the offsite deposition rate is adjusted by a factor of 0.44 (20 ft/45 ft). Based on these assumptions, the dose to an adolescent standing at very close range during application of 2,4-D at 2.2 lb/ac (240 mg/m^2) on a 20-foot

(6-meter) swath can be estimated. This dose would be equivalent to 0.032 mg/kg (240 mg/m^2 x .45 x .44 x .10 (dermal absorption) x 0.27 m^2/40 kg).

This dose can be considered extreme because it is about 10 times higher than the highest dose to a supervisor spending an entire day in the immediate vicinity of a treatment site. The supervisor 2,4-D dose, based on actual field measurements, would be 0.0024 mg/kg (9.8 ha x 2.2 kg/ha x 1.1 x 10^{-4} mg/kg/kg). In these analyses, dose estimates for adolescents in the vicinity of spray operations are based on the supervisor data as determined in field measurments.

2.4.6.3 General Population Oral Doses from Beef and Vegetables

Since cattle do not routinely graze on rights-of-way, herbicide dose to cattle will be greatly reduced. Work by Maybank et al. (1977) has shown that within 5 meters of a 13.7-meter wide spray swath, drift deposits on horizontal surfaces would be less than 1 percent of the concentration on the spray site. Thus, dose to cattle and subsequent dose to humans can be assumed to be 1 percent or less of the doses calculated for other projects being considered.

Oral dose from eating herbicide-contaminated vegetables is calculated using the same consumption rates as in scenarios for other projects. Vegetable gardens are assumed to be located 225 feet (60 meters) from the treatment area. Spray deposition factors for vegetation predicated on a 10-meter spray swath are provided in Table 2-6. Spray factors for a 60-meter distance are, therefore, multiplied by 1.2 (12 m/10 m) to account for the difference in width of the right-of-way treatment area. Oral dose for a 154-pound (70-kg) adult consuming 0.5 lbs (.23 kg) of vegetables daily is calculated on the assumption that no loss of herbicide occurs in washing or cooking. Vegetable consumption patterns (leafy and small fruit) assumed for open-range projects are also assumed for these projects. Worst-case daily dose based on an application rate of 1.2 kg/ha, is 2.5 x 10^{-4} mg/kg ((1.2 x 1.2 kg/ha x 0.1 mg/kg^2-ha x 0.10 kg/person x person/70 kg) + (1.2 x 1.2 kg/ha x 0.1 mg/kg^2-ha x 0.4 kg/person x person/70 kg x 0.05)).

As a worst-case approximation of the dose to a person who walks through the travelway shortly after treating the travelway, the average dose factors from Table 2-3 for a supervisor present during the entire treatment will be used. The dose from a 24-acre (9.8-ha) travelway project treated with picloram at 1.1 lbs/ac (1.2 kg/ha) would be 5.6 x 10^{-5} mg/kg (9.8 ha x 1.2 kg/ha x 4.8 x 10^{-6} mg/kg^2). This dose is equivalent to 0.056 ug/kg (microgram/kilogram).

2.4.6.4 General Population Dosage from Aquatic Contamination

As discussed in Section 2.1.4, many rights-of-way are located relatively close to stream channels. Herbicide applications could affect water quality through drift into the stream at the time of application and through runoff into the stream during subsequent rainstorms.

Table 2-31. Daily dosage to visitors and residents in the vicinity of right-of-way and riparian projects sprayed with 2,4-D, picloram, amitrole, dicamba, or glyphosate.

	2,4-D (microgram/[1/] kilogram/day)	Amitrole or Picloram (microgram/[1/] kilogram/day)	Glyphosate or Dicamba (microgram/[1/] kilogram/day)
Adult dermal dose	0.038	0.0019	0.019
Adolescent dermal dose	2.4	0.24	1.2
Infant dermal dose	0.09	0.0045	0.045
Adult/adolescent oral dose (beef)	0.0071	0.0071	0.0071
Infant oral dose (beef)	0.0083	0.0083	0.0083
Adult/adolescent oral dose (veg)	0.49	0.25	0.25
Infant oral dose (veg)	0.69	0.34	0.34
Visitor entry or walk along ROW	0.56	0.056	0.28
Adult oral dose (water)	5.7	2.8	2.8
Adolescent oral dose (water)	7.5	3.7	3.7
Infant oral dose (water)	8.3	4.2	4.2
Adult/adolescent oral dose (fish)	0.094	0.047	0.047
Infant oral dose (fish)	0.11	0.055	0.055

[1/] Micrograms (ug) are converted to milligrams by dividing micrograms by 1000.

Table 2-32. Daily dosage to visitors or residents in the vicinity of right-of-way and riparian projects sprayed with mixtures of 2,4-D/picloram or 2,4-D/dicamba.

	2,4-D/Picloram (microgram/ kilogram/day)	2,4-D/Dicamba (microgram/ kilogram/day)
Adult dermal dose	0.014/0.0005	0.014/0.0047
Adolescent dermal dose	0.9/0.06	0.9/0.3
Infant dermal dose	0.034/0.0011	0.034/0.011
Adult/adolescent oral dose (beef)	0.0071/0.0071	0.0071/0.0071
Infant oral dose (beef)	0.0083/0.0083	0.0083/0.0083
Adult/adolescent oral dose (veg)	0.185/0.062	0.185/0.062
Infant oral dose (veg)	0.26/0.086	0.26/0.086
Visitor entry or walk along ROW	0.21/0.01	0.21/0.07
Adult oral dose (water)	2.14/0.71	2.14/0.71
Adolescent oral dose (water)	2.81/0.94	2.81/0.94
Infant oral dose (water)	3.13/1.04	3.13/1.04
Adult/adolescent oral dose (fish)	0.035/0.012	0.035/0.012
Infant oral dose (fish)	0.041/0.014	0.041/0.014

Table 2-33. Daily dosage to visitors and residents in the vicinity of right-of-way and riparian projects sprayed with hexazinone, tebuthiuron, or atrazine.

	Tebuthiuron (microgram/ kilogram/day)	Hexazinone (microgram/ kilogram/day)	Atrazine (microgram/ kilogram/day)
Adult dermal dose	NA[1]	0.038	0.038
Adolescent dermal dose	NA[1]	2.4	1.2
Infant dermal dose	NA[1]	0.09	0.09
Adult/adolescent oral dose (beef)	0.071	0.0071	0.71
Infant oral dose (beef)	0.083	0.0083	0.83
Adult/adolescent oral dose (veg)	NA[1]	0.25	0.25
Infant oral dose (veg)	NA[1]	0.34	0.34
Visitor entry or walk along ROW	0.003	0.56	0.28
Adult oral dose (water)	2.8	2.8	2.8
Adolescent oral dose (water)	3.7	3.7	3.7
Infant oral dose (water)	4.17	4.17	4.17
Adult/adolescent oral dose (fish)	0.47	0.236	0.236
Infant oral dose (fish)	0.55	0.275	0.275

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[1] Tebuthiuron is applied as a pellet. Thus drift cannot contact members of the general public or off-site vegetation.

Table 2-34. Summary of references for herbicide concentrations in runoff.

Pesticide	References
2,4-D	Review by Norris 1981
Picloram	Davis and Ingebo 1973 Baur et al. 1972 Bovey et al. 1974 and 1975 Mayeux et al. 1984 Norris et al. 1982 Neary et al. 1985
Dicamba	Trichell et al. 1968 Schwab et al. 1973
Glyphosate	Edwards et al. 1980 Newton et al. 1984
Hexazinone	Bouchard et al. 1985 Neary et al. 1983
Amitrole	Marston et al. 1968 Norris 1968
Atrazine	Review in USDA Forest Service 1986a
Tebuthiuron	Review in USDA Forest Service 1984 Elanco 1983

In order to estimate the herbicide deposition in the water body adjacent to treatment areas, several assumptions were made. As discussed in Section 2.4.6.2, Maybank et al. (1977) reported numerous spray trials which determined spray deposition both on target and in close proximity off target under a variety of conditions. Maybank has shown that within 5 meters of a 14-meter-wide ground-rig spray swath, drift deposits on horizontal surfaces would be less than 1 percent of the nominal application rate on target. Assuming a stream averaging 3 feet (1 meter) wide, 4 inches deep, and flowing at 1 cubic foot (28.3 liters) per second (cfs), the drift deposition onto 8 kilometers (8,000 meters) of stream adjacent to the 8 kilometers of road being sprayed would total 10,400 mg (1.3 kg/ha x 0.01 x 1 m x 8,000 m x ha/10,000 m^2 x 1,000,000 mg/kg). The drift deposition would be diluted into the water that flowed past the project in the 6 hours (21,600 seconds) during which spraying occurred. Thus, the maximum concentration at any time would be 0.017 mg/liter (10,400 mg x 1 cfs/28.3 L x 1/21,600 sec).

Maximum instream concentrations from herbicide-contaminated runoff will be highly dependent on site-specific characteristics. Table 2-34 provides a listing of numerous studies that have measured herbicide runoff concentration adjacent to spray areas. The review articles listed in this table (such as Norris 1981) summarize available literature on a herbicide and thus incorporate data from numerous studies.

The literature review indicates several things. Even when runoff concentrations are measured at the edge of large application areas, maximum runoff concentrations are less than 1 mg/liter and typically less than 0.1 mg/liter (with adjustments made for application rates). These maximum concentrations occur for a very short period, typically during the first significant rainfall after application. These concentrations are the maximum that might occur adjacent to the project, for example, in a drainage ditch or culvert. Concentrations in stream water would be 10 to 100 times less because of dilution with the base flow of the stream. Thus, the maximum concentration in stream water would be 0.1 mg/liter or less.

As a check on the reasonableness of this concentration estimate, it is also possible to calculate instream concentrations based on the total quantity of herbicide that might be lost in runoff. Studies with picloram and hexazinone, the most mobile of the herbicides of interest, have shown that between 0.35 percent and 6.0 percent of the total applied herbicide is lost in runoff in time periods ranging from months to years (see Mayeux et al. 1984; Davis and Ingebo 1973; Norris et al. 1982; Bouchard et al. 1985; and Neary et al. 1983). Assuming, for example, that 2 percent of the applied herbicide was lost in runoff in a 24-hour period and none was degraded or adsorbed by sediments, the herbicide concentration in a 1 cfs stream adjacent to a 1.2 kg/ha application on a 9.8 hectare right-of-way project would be 0.096 mg/L (1.2 kg/ha x 9.8 ha x 0.02 x 1,000,000 mg/kg x 1 sec/28.3 liters x 1/86,400 sec).

A 70 kg adult who drank 2 liters of water in a day with a herbicide concentration of 0.1 mg/L would receive a dose of 0.0029 mg/kg (2 liters x 0.1 mg/liter x persons/70 kg). The doses to adolescents and infants assuming consumption of 1.5 liters and 0.5 liters of water, respectively, would be 0.0036 mg/kg and 0.0042 mg/kg, respectively.

An assumption of 2 percent loss in 24 hours will also be used when calculating maximum oral doses from application rates other than 1.2 kg/ha.

Aquatic organisms that are exposed to herbicides in water can absorb and retain some of the herbicide. Agriculture Handbook No. 633 (USDA Forest Service 1984) provides an extensive review of the environmental fate and toxicological literature for the herbicides of interest in these risk analyses. This review indicates that these herbicides have little tendency to bioaccumulate in the environment. Bioaccumulation factors of 1.0 or less are indicated for all herbicides of interest except atrazine and hexazinone which may have factors as high as 5.0 and tebuthiuron which may have a factor as high as 10 (USDA Forest Service 1986a). Assuming that the herbicide concentration in the 15 cfs fishery stream was diluted 15 times from the roadside stream concentration (15 cfs/1 cfs), and that the fish in the stream can absorb herbicide to their maximum bioaccumulative capacity very quickly, the atrazine or hexazinone concentration would be 0.033 m/kg (0.1 mg/L x 5.0 mg/kg/mg/L x 1/15). The concentrations of the herbicides with bioaccumulation factors of 1.0 or lower would be less than one-fifth these concentrations.

Worst-case daily oral dose to a 70 kg fisherman who catches and consumes 0.5 kg (1.1 pounds) of fish contaminated with atrazine or hexazinone would be 2.4 x 10^{-4} mg/kg (0.033 mg/kg x 0.5 kg x person/70 kg) or 0.24 ug/kg (microgram/kilogram). Adolescent and infant doses are based on assumed consumption of 0.3 kg and 0.1 kg of fish, respectively.

Actual doses from eating such fish would very probably be many times less since it typically takes many days of exposure to a given concentration of herbicide in water for a fish to bioaccumulate to the steady state maximum concentration indicated by the bioaccumulation factors. The maximum water concentrations used in these dose calculations would likely never occur or would occur at most for a very few hours.

2.4.7 Affected Population Exposure and Dose from Aerial Spray Projects

Relatively few herbicides are suitable for aerial application. This analysis examines the impacts of aerial applications of 2,4-D, 2,4-D/dicamba mixtures, picloram, and tebuthiuron pellets.

2.4.7.1 Worker Doses

Worker doses can be calculated for pilots, mixer/loaders, supervisors, and observers associated with aerial spray projects. Aerial application rates are assumed to be the same as the ground application rates (see Table 2-7).

As noted in Section 2.4.3.1, the various mixing and application errors discussed in Section 2.2 are assumed to be accounted in the worker dosage factors provided on Table 2-3. Thus, the typical application rates from Table 2-7 are used to calculate worker dose. For example, the average pilot dose from spraying 2,4-D at 2.2 kg/ha on a 49-ha (120-acre) project would be 0.017 mg/kg (1.6 x 10^{-4} mg/kg/kg x 49 ha x 2.2 kg/ha). Mixer/loader, supervisor, and observer dosage (both average and high) can be calculated in similar fashion using the appropriate factors from Table 2-3. Worker dosages are provided on Tables 2-35 and 2-36.

2.4.7.2 General Population Dermal Dose from Drift

General population doses from drift are calculated based on typical and worst-case assumptions as discussed in Section 2.4.2. In the worst-case scenario drift deposition at 200 meters is assumed to be 0.16 percent of the nominal application rate on-site. Thus, for a 1.2 kg/ha application of picloram the drift deposition would be 0.19 mg/m^2 (1.2 kg/ha x 0.0016 x 1,000,000 mg/kg x 1 ha/10,000 m^2). This drift deposition is only slightly higher than the drift estimate for large (500 acres) ground application projects and again indicates that the drift deposition levels estimated for ground application projects would be an extremely rare event.

As discussed in Section 2.4.2, the drift deposition under routine conditions is assumed to be 0.1 percent of worst-case levels or 0.00019 mg/kg (0.19 mg/m^2 x 0.001).

The same assumptions regarding exposed skin surface area and dermal absorption of herbicides are made for aerial projects as for ground projects in the previous sections. For example, the adult dose of picloram under worst-case conditions can be estimated as 1.0 x 10^{-5} mg/kg (0.19 mg/m^2 x 0.37 m^2 x adult/70 kg x 0.01) or 0.01 ug/kg. Dose estimates for adolescents and infants for both worst-case and routine scenarios are provided on Table 2-37 through 2-40. Because tebuthiuron would be applied as pellets, drift impacts would not be observed and corresponding doses are not provided. (Text continued on page 87).

Table 2-35. Dosage levels for pilots and mixer/loaders on aerial spray projects.

	Pilots		Mixer/Loaders	
	Average	High	Average	High
	(milligram/ kilogram/day)		(milligram/ kilogram/day)	
2,4-D	0.017	0.069	0.019	0.052
2,4-D/ Dicamba	0.006/ 0.002	0.025/ 0.009	0.007/ 0.003	0.019/ 0.007
Picloram	0.002	0.007	0.002	0.005
Tebuthiuron	0.0001	0.0003	0.0001	0.0003

Table 2-36. Dosage levels for supervisors and observers on aerial spray projects.

	Supervisors		Observers	
	Average	High	Average	High
	(milligram/ kilogram/day)		(milligram/ kilogram/day)	
2,4-D	0.003	0.010	0.0005	0.0014
2,4-D/	0.0009/	0.004/	0.0002/	0.0005/
Dicamba	0.0004	0.002	0.00007	0.0002
Picloram	0.0003	0.0012	0.00005	0.00014
Tebuthiuron	0.00003	0.00012	0.000005	0.00001

Table 2-37. Daily dosage to visitors and residents in the vicinity of projects aerially sprayed with 2,4-D.

	Worst Case (microgram/ kilogram/day)[1]	Routine (microgram/ kilogram/day)[1]
Adult dermal dose	0.20	0.0002
Adolescent dermal dose	0.26	0.0003
Infant dermal dose	0.48	0.0005
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	6.6	0.0066
Infant oral dose (veg)	10.1	0.0101
Adult oral dose (water)	34	0.68
Adolescent oral dose (water)	30	0.60
Infant oral dose (water)	50	1.0
Visitor entry to spray site	10	3.0
Oral dose/sprayed wild food	51	51

1/ Micrograms (ug) are converted to milligrams by dividing micrograms by 1,000.

Table 2-38. Daily dosage to visitors and residents in the vicinity of projects aerially sprayed with picloram.

	Worst Case (microgram/kilogram/day)[1]	Routine (microgram/kilogram/day)[1]
Adult dermal dose	0.010	0.00001
Adolescent dermal dose	0.013	0.00001
Infant dermal dose	0.024	0.00002
Adult/adolescent oral dose (beef)	0.71	0.71
Infant oral dose (beef)	0.83	0.83
Adult/adolescent oral dose (veg)	3.3	0.0033
Infant oral dose (veg)	5.1	0.0051
Adult oral dose (water)	17	0.34
Adolescent oral dose (water)	15	0.30
Infant oral dose (water)	25	0.50
Visitor entry to spray site	1.2	0.3
Oral dose/sprayed wild food	26	26

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[1]/ Micrograms (ug) are converted to milligrams by dividing micrograms by 1,000.

Table 2-39. Daily dosage to visitors and residents in the vicinity of projects aerially treated with tebuthiuron.

	Worst Case (microgram/[1] kilogram/day)	Routine (microgram/[1] kilogram/day)
Adult dermal dose	NA[2/]	NA[2/]
Adolescent dermal dose	NA[2/]	NA[2/]
Infant dermal dose	NA[2/]	NA[2/]
Adult/adolescent oral dose (beef)	7.1	7.1
Infant oral dose (beef)	8.3	8.3
Adult/adolescent oral dose (veg)	NA[2/]	NA[2/]
Infant oral dose (veg)	NA[2/]	NA[2/]
Adult oral dose (water)	17	NA[3/]
Adolescent oral dose (water)	15	NA[3/]
Infant oral dose (water)	25	NA[3/]
Visitor entry to spray site	0.12	0.03
Oral dose/sprayed wild food	NA[2/]	NA[2/]

[1/] Micrograms (ug) are converted to milligrams by dividing micrograms by 1,000.

[2/] Drift would not occur because tebuthiuron is applied in pellet form. Tebuthiuron pellets would not adhere to wild food and uptake of pelletized tebuthiuron in the roots of fruit-bearing shrubs and plants and other wild foods would prevent development.

[3/] Drift into streams would not occur because tebuthiuron is applied as pellets. Water concentrations are only calculated for the overflight scenario.

Table 2-40. Daily dosage to visitors and residents in the vicinity of projects aerially sprayed with 2,4-D/dicamba.

	Worst Case (microgram/kg/day)[1/] 2,4-D/Dicamba	Routine (microgram/kg/day)[1/] 2,4-D/Dicamba
Adult dermal dose	0.076/0.025	0.00008/0.00003
Adolescent dermal dose	0.097/0.032	0.0001/0.00003
Infant dermal dose	0.18/0.06	0.0002/0.00007
Adult/adolescent oral dose (beef)	0.71/0.71	0.71/0.71
Infant oral dose (beef)	0.83/0.83	0.83/0.83
Adult/adolescent oral dose (veg)	2.2/0.82	0.0022/0.00082
Infant oral dose (veg)	3.4/1.3	0.0034/0.0013
Adult oral dose (water)	12/4.2	0.24/0.084
Adolescent oral dose (water)	10/3.8	0.20/0.08
Infant oral dose (water)	17/6.2	0.34/0.124
Visitor entry to spray site	4.0/2.0	0.9/0.4
Oral dose/sprayed wild food	20/6.0	20/6.0

[1/] Micrograms (ug) are converted to milligrams by dividing micrograms by 1,000.

2.4.7.3 General Population Oral Doses, Beef and Vegetation

Oral doses to humans from eating cattle that grazed on herbicide-treated forage are estimated using the same assumptions outlined for general ground application projects. These dose values are provided on Tables 2-37 through 2-40.

Residents downwind of aerial spray projects could also receive oral doses from consuming drift-contaminated garden vegetables. Based on a review by Hoerger and Kenaga (1972), the residue on leafy vegetables is about 100 parts per million (mg/kg) for every pound/acre (1.1 kg/ha) of pesticide applied or deposited. Under worst-case conditions, the FSCBG model predicts that pesticide residues 200 meters from spray sites would be 0.16 percent of the residues on-site. On this basis, predicted pesticide residue on leafy vegetables 200 meters from a 1.2 kg/ha aerial application would be 0.19 mg/kg (1.2 kg/ha x 100 mg-ha/kg^2 x 0.0016). However, data developed by Yates et al. (1978) indicate that drift residue measured on wheatgrass could be about 10 times the levels predicted using the methodology outlined above. The difference may be due to the increased collection efficiency of the leafy vegetation. For this reason, pesticide residue on leafy vegetation that is 200 meters from an aerial project is assumed to be 1.9 mg/kg/under worst-case conditions and 0.0019 mg/kg under routine conditions (given a 1.2 kg/ha aerial application). As discussed in Section 2.4.2, residues in mg/kg on small fruits and vegetables are assumed to be 5 percent of the residues on leafy vegetables.

Food consumption patterns are assumed to be identical to those discussed with ground application projects (see, for example, Section 2.4.3.3). On this basis, the worst-case dose of picloram to an adult eating vegetables from a garden downwind of an aerial spray project (1.2 kg/ha) would be 3.3 x 10^{-3} mg/kg (((1.9 mg/kg x 0.1 kg) + (1.9 mg/kg x 0.4 kg x 0.05)) x (adult/70 kg)) or 3.3 ug/kg. Oral doses under worst-case and routine conditions are provided for adults, adolescents, and infants on Tables 2-37 through 2-40.

2.4.7.4 General Population, Oral Doses from Aquatic Contamination

Buffer zones of 50 feet (15 meters) to live water can eliminate most aquatic impacts associated with aerial spraying. Peak concentrations in overland flow are reduced from 70 to 90 percent within 15 meters of a spray plot (Asmussen et al. 1977 and Trichell et al. 1968). However, the possibility exists that spray drift in excess of levels estimated in Section 2.4.6.4 could impact the streams. Likewise the possibility exists that spray aircraft could accidentally spray the stream directly. This section will examine the impacts of both scenarios.

As a worst case, an aircraft is assumed to directly spray 1,000 meters (yards) of stream. The stream is assumed to be 3 feet (almost 1 meter) wide, 4 inches (0.1 meter) deep, and flow at 1 cubic foot per second. The concentration in the stream immediately after a 1.2 kg/ha (120 mg/m^2) application would be 1.2 mg/liter (120 mg/m^2 x 1/0.1 m x 1 m^3/1,000 liters).

This concentration in the stream would begin to fall immediately after application because of dilution. Assuming that an adult were able to drink a liter (about a quart) of this water immediately after spraying, his dose would be 0.017 mg/kg (1.2 mg/liter x 1 liter x adult/70 kg) or 17 ug/kg (microgram/kilogram). Adolescent and infant doses are calculated on the assumption that one-half and one-quarter liters are consumed respectively. These worst-case dose estimates from the consumption of water contaminated during a direct spray incident are provided on Tables 2-37 through 2-40.

Absorption of herbicides by fish is not reasonably expected because this brief contamination would be diluted quickly as it moved downstream.

Drift contamination to surface water can also be calculated for conditions that could occasionally occur at a spray site. These conditions are assumed to include 10-foot release height and 6-mph winds. Assuming the edge of the treatment area is 50 feet (15 meters) upwind of the stream and that drift deposition is 2 percent of the concentration on-site (see Figure 11 of USDA Forest Service 1984a), the stream surface deposition from a 1.2 kg/ha (120 mg/m^2) would be 2.4 mg/m^2 (0.02 x 120 mg/m^2). If the stream depth averaged 4 inches (0.1 meters), the herbicide concentration in the stream would be 0.024 mg/l (2.4 mg/m^2 x 1/0.1 m x 1 m^3/1,000 liters). Maximum doses to human consumers of this water would be about 2 percent of the doses calculated under the worst-case direct spray scenario.

2.4.7.5 Visitor Doses

The dosage of persons reentering a spray site and the dosage to persons eating wild foods are estimated using the methods outlined in Section 2.4.3.4. These dose estimates are provided on Tables 2-37 through 2-40.

2.5 REVIEW OF GENERAL TOXICITY DATA FOR HERBICIDES

The significance of the dose levels developed in Sections 2.1 through 2.4 is determined in part by comparison to dose levels that produce systemic toxic effects in laboratory animals. As discussed in Section 1.8, Forest Service Handbook No. 633 reviews toxicity and environmental fate of these herbicides. Data in this handbook, as well as more recent data from the U.S. EPA pesticide registration review process, form the basis of this review of toxicity data.

The toxic effects of a compound can be measured on any number of animal species using a variety of experimental protocol. The acute toxicity of a chemical compound is often indicated by the one-time or short-term dose that is lethal to 50 percent of a group of treated animals. This value is abbreviated as the LD_{50} and is expressed as the mass unit of compound (usually in grams, milligrams, or micrograms) administered per mass unit of organism (usually in kilograms). It follows that the higher the LD_{50} value, the less toxic the compound.

LD_{50} values will vary among species tested. Because there is no universally accepted method for determining which animal species would provide the most suitable model for effects on man, the LD_{50} value for the species most sensitive to a particular herbicide is reported in Table 2-41. These values are based on a review of herbicide toxicological data provided by Agriculture Handbook No. 633 (USDA Forest Service 1984 and 1986a).

Table 2-41. Summary of acute toxicity thresholds based on results from the most sensitive species.

Herbicide	Acute oral LD_{50} in milligram/kilogram [1]
Amitrole	1,100
Atrazine	1,400
2,4-D	100
Dicamba	566
Glyphosate	3,800
Hexazinone	860
Picloram	2,000
Tebuthiuron	644

[1] Based on review in Agriculture Handbook No. 633 (USDA Forest Service 1984 and 1986a).

Lethality represents a rather extreme benchmark for judging possible effects from the use of herbicides. A variety of significant health effects could occur in an organism at doses less than the lethal dose. Therefore, policies regarding the acceptable intake levels for chemical compounds are most often based on toxicity tests designed to find the dose level that produces no effects in the animal species tested. This dose is referred to as the no-observed effect level (NOEL). The NOEL is the highest dose level tested that does not affect the organism's health or well being over the test duration. A NOEL can be determined for acute (single dose or short term), subchronic (generally 30- to 90-day dosing studies), and chronic tests of a compound. Once again, the higher the NOEL value the less toxic the compound.

Chronic and subchronic tests can be designed to reveal general systemic effects such as effects on kidney and liver function, blood chemistry, growth rate, and other measures of health. More specialized tests can also be designed to investigate selected systems. For example, the effects of chemicals on human reproductive systems and on fetuses (fetotoxicity) is a concern. To test for these effects, multi-generation feeding studies are conducted. These tests involve dosing animals and their offspring for several generations to test for effects on rate of reproduction as well as maternal and offspring health. Pregnant animals can also be dosed at higher rates for a shorter period of time to test for teratogenic effects (formation of gross abnormalities such as limb malformations) or general fetotoxic effects such as reduced offspring weight.

As noted in the previous paragraphs, chronic NOEL values are generally determined in tests of laboratory animals. Although establishing a NOEL in animal feeding studies is a relatively straightforward, albeit expensive process, extrapolating these findings to humans is more controversial. The U.S. EPA faces this issue when deciding how much pesticide to allow in foods and that agency's approach to the problem illistrates one solution.

In registering pesticides for use on agricultural commodities for human consumption or on feed for animals subject to human consumption, the U.S. EPA establishes tolerances (allowable amounts) for residues of the pesticide. These tolerance levels are based on the toxicity data establishing NOEL's for the pesticide and a projection of human consumption patterns. The U.S. EPA uses the NOEL from the chronic dose studies with the species that is most sensitive to the compound as a basis for calculating an acceptable daily intake (ADI) for the compound. An acceptable intake quantity can then be apportioned among various crops for which residue tolerances are requested.

The time-honored approach for establishing an ADI or a safe level of herbicide dose is to divide the threshold dose or NOEL established from chronic animal studies by a "safety factor" (Doull et al. 1980 and NAS-NRC 1977). The safety factors are needed to account for the differences in duration of exposure, absorption, metabolism, and excretion between humans and test animals. For example, on a body-weight basis, humans can be more vulnerable to drugs than are experimental animals by a factor of 6-12 (NAS-NRC 1977). In other words, if a dose of 10 mg/kg/day elicits a response in animals, a dose of 1 to 2 mg/kg/day may elicit the same response in humans. Often if the dose is scaled on a surface area basis, this increased vulnerability disappears.

In addition to accounting for differences between animal species and humans (interspecies differences), the safety factor should also account for the differences among humans (intraspecies differences). For example, in setting the ADI for atrazine, the U.S. EPA divided the NOEL from the chronic dog study (1.5 mg/kg/day) by a safety factor of 100. This safety factor of 100 can be considered to include a tenfold safety factor to account for the difference between animal species and humans and an additional tenfold safety factor to account for sensitive humans.

In discussing ADI's, a final point should be emphasized. The ADI assumes that the person will be dosed every day for a lifetime. Larger doses can be tolerated for shorter periods of time without effect. As is emphasized below, possible exposures resulting from Forest Service spraying would occur only a few days per year.

The following sections provide test results upon which NOEL and ADI factors are based. Carcinogenic and mutagenic effects are discussed separately in Section 2.7.

2.5.1 Amitrole Threshold Effects

In a 13-week subchronic rat-feeding study, thyroid iodine uptake was reduced and enlarged thyroids were observed at feeding rates as low as 2 ppm in the diet. Significant functional changes occurred after only 1 week of feeding (U.S. EPA 1985b). A dose of 2 ppm is equivalent to 0.01 mg/kg (milligrams of pesticide dose per kilogram of animal body weight). Conversion factors relating ppm in food to mg/kg body weight are contained on page 35 of Agriculture Handbook 633 (USDA Forest Service 1984). A NOEL of 0.5 ppm (0.025 mg/kg/day) was reported in this study.

No teratogenic effects were observed in the offspring of rats dosed with amitrole at 20 and 100 mg/kg/day. However, in a two-generation reproductive study, hyperplasia of the thyroid was observed at all doses (25 ppm and higher). Runting was observed in offspring of parents fed 500 and 1,000 ppm.

An ADI for amitrole has not been set because the herbicide is not approved for use on crops or forage.

2.5.2 Atrazine Threshold Effects

Ciba-Geigy submitted the results of a 2-year rat-feeding study to the U.S. EPA in 1986 (USDA Forest Service, 1986). A NOEL of 70 ppm (parts per million in food) was indicated in this study. This does is equivalent to 3.5 mg/kg based on the food-to-body-weight factors for rats (see amitrole discussion). At doses above the NOEL, the symptoms in dosed rats included reduced body weight, reduced hematocrit (red blood cell count), and reduced hemoglobin. The results of the rat feeding are very similar to a 2-year dog-feeding study that established a NOEL of 150 ppm (equivalent to a NOEL of 3.7 mg/kg/day, based on the food-to-body-weight factors for dogs.) At doses above the NOEL, decreased body weight and reduced hemoglobin and hematocrit values were also observed (USDA Forest Service 1986b).

A three-generation rat-reproduction study reported no reproductive or systemic effects at the highest dose tested (100 ppm or 5 mg/kg/day). No teratogenic effects (birth defects) were observed in rats at doses up to 1,000 mg/kg/ (highest dose tested). In the same study, fetotoxic and maternal toxicity NOEL's were established at 100 mg/kg/day (USDA Forest Service 1987b). An ADI of 0.037 mg/kg has been set by EPA.

2.5.3 2,4-D Threshold Effects

Since its commercial introduction in the 1940's, 2,4-D has been the subject of numerous animal-dose studies, clinical reports, and human epidemiology studies (the comparative study of disease rates in subsets of the human population). In 1980, the EPA formally reviewed the toxicology information available on 2,4-D. Based on this review the EPA required manufacturers of 2,4-D to submit new data on the following potential health effects: acute toxicity (oral and dermal), oncogenicity (tumor formation) in rat and mouse, reproduction, teratogenicity (birth defects), neurotoxicity, and metabolism. All required studies have been submitted and the results of these studies are summarized in the Pesticide Fact Sheet on 2,4-D (U.S. EPA, 1987).

In a teratology (birth defect) study, teratogenic effects were not observed in rats at the highest dose tested (75 mg/kg/day). The fetotoxic NOEL was 25 mg/kg/day. At 75 mg/kg/day, a delay in ossification (hardening) of the bones was observed in offspring. In a multi-generational rat-reproduction study, a fetotoxic NOEL of 5 mg/kg/day was seen. At a dose of 20 mg/kg/day, a decrease in maternal and pup body weight was observed.

The lowest NOEL reported for 2,4-D was observed in a rat-feeding study. The 1-year report for this study indicated a NOEL of 1 mg/kg/day based on kidney effects at higher dose levels. An ADI of 0.01 mg/kg/day has been based on this NOEL.

The medical literature reports several cases of peripheral neuropathy apparently resulting from exposure to 2,4-D. Peripheral neuropathy is the disruption of the nervous system characterized by some or all of the following:

numbness in hands and feet,
loss of balance,
aching in extremities,
fatigue,
nausea.

Recovery in some cases is very prolonged and may not be complete even after several years.

Although many of the reported cases of peripheral neuropathy occurred after massive doses, effects in some people have been noted at much lower apparent doses. The conclusions of Berkley and Magee (1963) are appropriate.

> "Despite the extensive use of 2,4-D preparations, resultant peripheral neuropathy is very rare, and an affected individual probably has some predisposition to neuropathy or susceptibility to the toxin. Nevertheless, as it cannot be determined who is predisposed or susceptible, and as no antidote to 2,4-D intoxication is known, prevention is simpler than treatment."

As reported in the Pesticide Fact Sheet for 2,4-D, neurotoxicity has not been induced in animal studies.

2.5.4 Dicamba Threshold Effects

The test data upon which many of the tolerance determinations for dicamba are based require replacement. The Federal Register (March 16, 1983, pg. 11119) reports a 2-year dog-feeding study with dicamba with a NOEL of 1.25 mg/kg/day. However, EPA has invalidated this study and substituted a 90-day rat-feeding study with a NOEL of 25 mg/kg/day for general toxic effects (U.S. EPA 1986a).

In a 2-year rat study, a systemic NOEL of greater than 2,500 ppm (125 mg/kg body weight), the highest dose tested, was established (EPA 1986b). Fetotoxic and maternal toxic effects have been observed in laboratory animals exposed to dicamba. A fetotoxic NOEL of 0.5 mg/kg was reported for a rabbit teratology pilot study, with resorptions reported at 1.0 mg/kg (EPA 1984c). A second rabbit teratology study resulted in a maternal NOEL and a fetotoxic NOEL of 3.0 mg/kg (EPA 1984c). Recent information from EPA (1985e) has placed the reproductive NOEL of dicamba at 2.5 mg/kg.

As indicated above, significant variability exists in the toxicity data base for dicamba. EPA based its tolerances on an ADI of 0.012 mg/kg/day. Although originally based on a NOEL of 1.25 mg/kg/day, the ADI could also be supported with the subchronic NOEL of 25 mg/kg/day using a higher safety factor (e.g., 2,000, see U.S. EPA 1986a).

2.5.5 Glyphosate Threshold Effects

A 26-month rat-feeding study produced no observable effects at the highest dose tested (30 mg/kg/day). A three-generation rat-reproductive study established a NOEL of 10 mg/kg/day. This NOEL is based on renal tubular dilation in the kidneys of the pups. No teratogenic effects were detected in teratology studies with glyphosate in rats and rabbits at the highest doses tested (1,000 and 350 mg/kg/day, respectively). Based on the three-generation rat-reproductive study, an ADI of 0.1 mg/kg/day has been established.

2.5.6 Hexazinone Threshold Effects

A NOEL of 200 ppm was determined in a 2-year mouse-feeding study (equivalent to 30 mg/kg/day) (EPA 1983b). A 2-year rat-feeding study provided a NOEL of 200 ppm (10 mg/kg/day) (EPA 1983b). The toxic effects observed during the mouse study were increases in liver size, a localized increase in liver cells, and localized tissue degeneration at the lowest effect level of 2,500 ppm (375 mg/kg/day).

A three-generation reproduction study established a NOEL of 1,000 ppm (50 mg/kg/day) in rats. A rabbit teratology study reported both a teratogenic and fetotoxic NOEL greater than 125 mg/kg (highest dose tested).

The most recent tolerances for hexazinone were issued in 1983 and are based on a NOEL of 25 mg/kg/day from a 90-day dog-feeding study. The ADI of 0.0125 mg/kg/day was calculated by dividing the NOEL by a safety factor of 2,000. The issuances of new tolerances will require completion of a chronic-feeding study with dogs. Thus, even though hexazinone has been reregistered, additional data are now required.

2.5.7 Picloram Threshold Effects

The California Department of Food and Agriculture reports the results of a recently completed 2-year rat-feeding study (CDFA 1986). At 12 months, adverse chronic effects were seen in the livers of rats fed at 60 and 200 mg/kg/day, but not at 20 mg/kg/day. It is interesting to note that these liver effects were not seen in the high-dose groups at the end of the 2-year study. Despite this evidence of possible increased tolerance to picloram doses with time, a NOEL of 20 mg/kg/day is derived from this test.

The Dow Chemical Company also reports the results of a 6-month feeding study with dogs. A NOEL of 7 mg/kg/day was reported based on decreased body weight, food consumption, and liver weight at higher doses (Dow undated). A 1-year dog study was completed recently and a final study report will soon be submitted to EPA.

Teratogenicity/fetotoxicity and reproductive function tests that are available for picloram all indicate low toxicity (USDA Forest Service 1984). Teratogenic or fetotoxic effects were not seen at doses up to 500 mg/kg/day. A three-generation rat-reproduction study provided a NOEL of 1,000 parts per million in food (50 mg/kg/day on a body weight basis).

The current ADI for picloram was calculated by dividing a 90-day dog-feeding study NOEL of 50 mg/kg/day by a safety factor of 2,000. The recent NOEL data reported in this analysis will eventually be incorporated into the registration process and may result in a higher ADI since less extreme safety factors would be required.

2.5.8 Tebuthiuron Threshold Effects

A 2-year rat-feeding study resulted in a NOEL of 400 ppm (20 mg/kg) (U.S. EPA 1986c). A 90-day dog-feeding study is also reported with a NOEL of 12.5 mg/kg. However, the dog study was not used as a basis for calculating an ADI for tebuthiuron. Rat and rabbit teratology studies produced no effects at the highest doses tested (90 and 25 mg/kg, respectively) (U.S. EPA 1983c). A two-generation rat-reproduction study provided a NOEL of 20 mg/kg/day (U.S. EPA 1983c).

The tebuthiuron ADI is based on the NOEL from the chronic rat-feeding study (20 mg/kg/day) and a safety factor of 100 (U.S. EPA 1983c). The ADI was not based on the lower NOEL from the 90-day dog-feeding study.

Table 2-42. Reproductive and general effect NOEL's and acceptable daily intake (ADI) values.

Herbicide	Reproductive effects in mg/kg/day [1]	General effects NOEL in mg/kg/day	ADI values in mg/kg/day and ug/kg/day [1]	Reference for ADI
Amitrole	5	0.025	NA [2]	
Atrazine	15	3.5	0.0375	Fed. Register 1/30/81 p. 63085
2,4-D	25	1.0	0.01 10	U.S. EPA 1985a
Dicamba	2.5	25	0.0125 12.5	Fed. Register 3/16/83 p. 11119
Glyphosate	10	10	0.1 100	Fed. Register 10/30/85 p. 45121
Hexazinone	50	10	0.0125 12.5	Fed. Register 8/17/83 p. 37214
Picloram	500	7	0.0250 25	Fed. Register 9/22/82 p. 41770
Tebuthiuron	20	12.5	0.2 200	Fed. Register 6/1/83 p. 24396

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[1] Doses are provided in milligrams of chemical dose per kilogram body weight per day (mg/kg/day). ADI values are also provided in micrograms (ug) to allow easier comparison with general population doses.

[2] The U.S. EPA has not established an ADI for amitrole because it has not approved its use on agricultural commodities.

2.5.9 The Utility of ADI's and NOEL's

Section 2.6 compares the dose values determined in Section 2.4 with the ADI's and NOEL's and discusses the significance of these comparisons. The remainder of Section 2.5 discusses the general toxicity of herbidies as formulated, including manufacturing by-products. The carcinogenic and mutagenic activity of herbicides is considered separately in Section 2.7.

2.5.10 Toxicity of Pesticide Formulations

As formulated for field use, pesticide active ingredients are mixed with a variety of compounds typically listed as "inert ingredients" on the label. These ingredients comprise various surfactants, adjuvants, and emulsifiers as needed to increase the usefulness of the pesticide. The chemical identities of these compounds are closely guarded trade secrets.

Environmental Protection Agency requires some toxicity testing of the formulated pesticides to indicate possible human health and environmental impacts of the formulations. Five mammalian tests are required for registration: acute oral LD_{50}, dermal sensitization, eye irritation, dermal LD_{50}, and acute inhalation. Agriculture Handbook No. 633 (USDA, Forest Service 1984 and 1986a) provides a review of these tests.

Table 2-43 compares the acute oral toxicity of the pesticide active ingredient and the acute oral toxicity of formulations of the pesticide. As indicated by this table, formulated mixtures are less toxic than the unformulated pesticide active ingredient, that is the LD_{50}s are higher.

Concern has also been expressed about the possible effect of surfactants in herbicide formulations on the absorption of herbicide active ingredients through human skin and the subsequent toxic effects of the herbicides. As indicated on Table 2-44, acute dermal toxicities of the active ingredient and formulated products are similar.

On some toxicity variables, differences are noted between formulations. For example, the Roundup formulation of glyphosate has much higher toxicity to fish than the Rodeo formulation (LC_{50} for trout of 11 parts per million (ppm) for Roundup and greater than 1,000 ppm for Rodeo). These differences are functions of the difference in the toxicity of the surfactants used in the formulation.

Table 2-43. Comparison of the acute oral toxicity of pesticide active ingredient and pesticide formulation.

Pesticide active ingredient	Oral LD$_{50}$ (mg/kg)	Formulation	Oral LD$_{50}$ (mg/kg)
Amitrole	1,100	Amitrol-T	5,000
Atrazine	1,400	AAtrex	1,750
		Atrazine, 80W	5,100
2,4-D (acid)	100	Dow Formulated 2,4-D	2,830
Dicamba	566	Banvel Technical	1,707
		Banvel DMA	1,028
Glyphosate	3,800	Roundup	5,400
		Rodeo	5,000
Hexazinone	860	Hexazinone (66% wettable powder)	4,495
		Hexazinone (10% Gridball)	7,500
		Hexazinone (20% Gridball)	5,000
Picloram	2,000	Tordon 22K (potassium salt)	10,300
		Picloram (isooctyl ester)	2,830
Tebuthiuron	644	Graslan 20P	2,000

Table 2-44. Comparison of the acute dermal toxicity of pesticide active ingredient and pesticide formulation.

Pesticide active ingredient	Dermal Oral LD_{50} (mg/kg)	Formulation	Dermal Oral LD_{50} (mg/kg)
Amitrole	>2,500	Amizol	10,000
Atrazine	---	AAtrex	9,300
2,4-D (acid)	1,400	2,4-D Dow formulated	3,980
Dicamba	>2,500	Banvel Technical	>2,000
Glyphosate	>7,940	Roundup	>7,900
		Rodeo	5,000
Hexazinone (90%)	>5,278	Hexazinone (66% wettable powder)	7,500
Picloram	>4,000	Tordon 22K (potassium salt)	>2,000
Tebuthiuron	>200	Tebuthiuron (60% pellets)	>1,200

The U.S. EPA has recently published two lists of inert ingredients of toxicological concern (Federal Register, April 22, 1987 p. 13305). List 1 contains about 55 chemicals or groups of chemicals shown to be carcinogens, developmental or reproductive toxicants, neurotoxins, or potential ecological hazards meriting the highest priority for regulatory action. List 2 contains about 60 chemicals or chemical classes with data suggestive of toxicity concerns or chemical structures similar to List 1 chemicals.

Some formulations of 2,4-D contain kerosene, a member of the chemical class "petroleum hydrocarbons" on List 2. The Draft Environmental Impact Statement for Vegetation Management in the Coastal Plain/Piedmont, as prepared by the Southern Region of the Forest Service, contains a risk assessment of the use of formulations containing kerosene. This review of the toxic characteristics of kerosene indicates a general systemic NOEL of 28 mg/kg/day and a reproductive effects NOEL of 751 mg/kg/day.

2.5.11 Toxicity of Herbicide Product Impurities

2.5.11.1 Dioxins and Phenolics in 2,4-D

The issue of contaminants in herbicides has received much publicity. The most noted case is the incidence of "dioxins," particularly 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) in 2,4,5-T (a discontinued herbicide). Because related compounds have been discovered in 2,4-D, concern has been raised over the possible health effects of herbicide impurities of these compounds.

Some confusion over possible health effects of herbicide impurities arises from mistaken terminology, particularly use of the terms "dioxin" and "TCDD." A brief digression in chemical nomenclature is warranted. The term "dioxin" is often used to refer to a class of compounds more properly referred to as dibenzo-p-dioxins. From a toxicological standpoint, the dibenzo-p-dioxins of most interest are those which have chlorine attached to one or more of the eight unsaturated carbon positions on the molecule. These compounds can be referred to as chlorodibenzo-p-dioxins. The number and location of chlorine molecules will greatly affect the toxicity of the compound. The most infamous and toxic of the chlorodibenzo-p-dioxins is the 2,3,7,8-tetrachloro-dibenzo-p-dioxin (2,3,7,8-TCDD), a compound with four chlorine atoms located at positions 2,3,7, and 8 on the molecule. Unfortunately all dioxins are often assumed to be similar or identical to 2,3,7,8-TCDD even though each of the approximately 75 chlorodibenzo-p-dioxin compounds varies significantly in its chemical and biological properties.

In the production of 2,4-D, an intermediate product is 2,4-dichlorophenol (2,4-DCP). Under some circumstances it is possible to join two molecules of 2,4-DCP to form the two-chlorine compound 2,7-dichlorodibenzo-p-dioxin (2,7-DCDD), a compound which differs only slightly in structure from 2,3,7,8-TCDD, but is over a millionfold less toxic. 2,7-DCDD was found in three of 30 samples of U.S.-produced 2,4-D along with traces of other relatively nontoxic chlorodioxins with three and four chlorines. The concentrations in the three positive samples ranged from 25 to 60 parts per billion (ppb).

If it were assumed, in the extreme, that all 2,4-D contained 100 ppb 2,7-DCDD, the maximum dose of 2,7-DCDD can be calculated to various exposed individuals. For example, if the maximum expected worker dose of 2,4-D is 0.3 mg/kg, the maximum dose of 2,7-DCDD to the exposed human would be 0.00000003 mg/kg.

Several toxicologic studies of 2,4-DCDD have been reported. Khera and Ruddick (1973) fed DCDD at dosages of 1 and 2 mg/kg daily to determine whether 2,7-DCDD could cause birth defects. The observed effect at 1 mg/kg was a modest degeneration of heart muscle fibers and some fluid accumulation around the heart in a few of the animals. A somewhat greater number of animals were affected at 2 mg/kg. Both effects are in the category of general fetal toxicity. No teratogenic effect was found. The 1 mg/kg dose is about 30 million times greater than the worst-case dose to workers in the previous paragraph.

The National Cancer Institute (1979) work was carried out by feeding 2,7-DCDD as 0.5 and 1 percent of the total diet for two years. The data indicated a "suggested" carcinogenic effect in male mice only that was not strong enough to support a conclusion that DCDD is a carcinogen. Female mice and rats of both sexes did not significantly respond. As will be discussed in Section 2.6, the dioxin 2,7-DCDD shows less carcinogenic potential than 2,4-D.

Additional concerns have been raised with the presence of 2,4-dichloro-phenol (2,4-DCP) in 2,4-D. As discussed above, 2,4-DCP is an intermediate product from which 2,4-D is synthesized. The eight manufacturers of 2,4-D in the United States have analyzed their products for 2,4-DCP. Total chlorophenols, of which 2,4-DCP is predominant, comprise about 0.3 percent of the product in the most contaminated sample. Other chlorophenols include 2,6-DCP and 2-chlorophenol and 4-chlorophenol, all of which are minor contributors. Many products contained no detectable chlorophenols.

The compound 2,4-DCP and other chlorophenols have very high vapor pressures and thus evaporate quickly. Although 2,4-DCP is one breakdown product of 2,4-D, microbial degradation of chlorophenols occurs much more quickly than degradation of 2,4-D and thus very little chlorophenol would accumulate in the environment (Vershueren 1983). Chlorophenols are also naturally occurring compounds. For example, 2,6-DCP is a pheromone of the lone star tick, Amblyomma americanum. The toxicity of 2,4-DCP is extremely low. Chronic (6 months) treatment of mice, at 0.1 percent (1,000 ppm) of total diet, produced no effects other than a slight liver enlargement (U.S. EPA 1980). The lethal dose is on the order of 10 times greater than that of 2,4-D.

2.5.11.2 Nitrosamine Formation from Glyphosate

The reaction of secondary amines such as glyphosate with nitrite ions to form various nitrosamines has received much attention. Testing has shown as many as 70 to 80 percent of nitrosamines are carcinogenic in animal tests. The formation of N-nitrosoglyphosate (NNG) has been documented (Khan and Young 1977). However, NNG levels in formulated herbicide products are less than 0.1 part per million and NNG has not been detected in raw agricultural products (U.S. EPA 1978, 1985c).

An additional concern has been the possible formation of NNG inside the human body through the reaction of nitrites in saliva and stomach fluids with glyphosate. Monsanto has reported tests of the carcinogenicity of NNG compound in mammals (Monsanto 1984). However, the validity of these tests by Industrial Bio-Test (IBT) is questionable due to numerous testing irregularities (U.S. EPA 1985d).

Although direct tests of NNG are not available, the indirect testing of possible health effects of NNG is accomplished through animal feeding studies. Rats, in particular, would be prone to in vivo nitrosamine formation because their stomach pH of 2.5 to 3.5 is more conducive to nitrosamine formation than that of humans whose stomach pH is in the range of 1.5 as demonstrated in a discussion of the kinetics of nitrosamine formation by Mirvish (1975).

The carcinogenic potential of glyphosate and NNG based on high-dose feeding studies is discussed further in Section 2.7.

2.6 ANALYSIS OF THE RISK OF THRESHOLD EFFECTS

In this section, the dose to hypothetical, maximum-exposed individuals is compared to the NOEL and ADI values for the herbicide in question. The maximum-exposed resident near general open-range and forest projects is assumed to receive a direct dose from drifting herbicides as well as an oral dose from the consumption of contaminated vegetables and beef. The maximum-exposed residents in the right-of-way and riparian scenario is assumed to receive a direct dose from drift and oral doses from drinking 2 liters of contaminated water and eating contaminated vegetables and fish (or beef). Consumption of beef instead of fish would lower the overall dose (see Tables 2-31, 2-32, and 2-33).

The maximum-exposed visitor to an open-range or forest project is assumed to spend the day on site and to eat contaminated wild food. It will also be noted that the maximum-exposed visitor could be a resident near the site. However, because the possible dose to a visitor from eating wild food from a spray site could be 10 to more than 100 times higher than any resident dose, the visitor scenario will be discussed separately.

There exists a small possibility that a worker would not only be exposed to worst-case levels on the job, but would also live near a spray site and be dosed through consumption of drift-contaminated vegetables or beef. However, even with the considerable overestimation of the exposure and dose levels in these residential dose pathways, the incremental impact on a worker would be negligible. For example, the high dose to a worker using recommended protection on a mid-sized 2,4-D project would be raised from 0.084 mg/kg/day to 0.086 mg/kg/day if the worker also ate contaminated beef and vegetables as a resident near such projects.

Tables 2-45 through 2-95 provide comparisons of the dose estimate for workers, maximum-exposed residents, and visitors with the NOEL values and the ADI values for each herbicide. The entry in each matrix element is the number by which a dose would have to be multiplied in order to equal the NOEL. For example, the table entry factor of 581 for an adult resident in the vicinity of a small, project sprayed with 2,4-D (see Table 2-47) was calculated by dividing the NOEL value for 2,4-D (from Table 2-41) by the sum of the adult dermal and oral doses from Table 2-9 (1,000 ug/kg/day)/((0.05 + 0.71 + 0.96) ug/kg/day)).

The NOEL/dose and ADI/dose comparisons provided on Tables 2-45 through 2-95 are for the day of maximum exposure which is generally the day of spraying. Since the direct dose from drift will only occur on the day of spraying, the comparison factors for subsequent days involving only oral doses would be higher, though often only slightly. In those cases where there is a marked contrast between dose on the first day and carry-over doses on subsequent days, two separate entries on the dose comparison tables are made. For example, the hypothetical adolescent who wanders very close to the spray site would receive a relatively high direct dose for a short period of time on the day of spraying. Maximum indirect doses on subsequent days would be much less. Thus, Tables 2-82 through 2-86 contain an entry that represents the safety factor for dermal and oral doses to this adolescent on the first day and a separate entry for doses on subsequent days. Separate entries are also made on these tables for visitors who merely enter sprayed areas and for visitors who both enter a sprayed area and consume wild foods that have been sprayed.

2.6.1 Discussion of ADI and NOEL Comparisons for the General Population Doses

In reviewing the NOEL/dose and ADI/dose comparisons on Tables 2-47 through 2-95, several patterns are noteworthy. With but few exceptions discussed below, the worst-case doses to maximum-exposed members of the general population are all below ADI values. The ADI presumes a daily dose everyday for a lifetime. As is discussed below, higher short-term doses can often be tolerated safely.

Amitrole use provides the most potential for adverse human impacts. Amitrole is a potentially potent antithyroid agent as evidenced by the low subchronic feeding levels (2 ppm in the diet or 0.1 mg/kg by body weight in rats) that result in significant effects on thyroid function. Under some circumstances doses to the general population could approach the NOEL for amitrole (see Tables 2-53 and 2-65). Doses to maximum-exposed residents could be 12 to 20 times lower than the NOEL for amitrole based on thyroid effects. Doses to visitors to a spray site who eat wild foods sprayed with amitrole could exceed the NOEL.

As discussed in Section 2.5, on a body-weight basis, humans can be as much as 6 to 12 times more sensitive to effects of chemicals than smaller test animals. Since the antithyroid effects of amitrole can be exhibited in a relatively short time (less than 90 days dose), the general population would be at risk from worst-case doses.

Several mitigating measures can greatly reduce the possibility of adverse impacts on the general public. Ensuring that cattle do not graze in treated areas will reduce the possibility of secondary doses to humans consuming beef. Posting amitrole-sprayed areas to warn the public against consuming wild foods on-site will reduce the possibility of these oral doses. Ensuring that spray sites are at least one-half mile from all residences, food crops, and gardens will ensure that drift-related doses are less than 5 percent of those doses calculated for the scenarios presented here. Careful site-specific analysis would be needed for any proposal to use this herbicide. At this time use of this compound in the Northern Region is unlikely.

The NOEL/dose and ADI/dose comparison for the remaining herbicides show that the exposure with the highest risk to the general public would involve consumption of wild food from a spray site. The dose comparisons show that a visitor to National Forest System lands can receive a dose that exceeds the ADI's if he or she collects and consumes a large quantity of sprayed, unwashed vegetation. For numerous reasons, there is a very low probability of this event. Very little land would actually be sprayed for noxious weeds (less than 0.04 percent of National Forest System land per year in the Northern Region). The targeted vegetation is not edible and berry bushes and other prime food-gathering areas generally do not occupy the same habitats that are infested with noxious weeds or poisonous plants. Finally, the appearance, odor, and taste of the sprayed vegetation would significantly reduce palatability of wild foods. Nonetheless, the calculated worst-case doses indicate that even if these improbable events were realized, the dose would be from 20 to 1,200 times less than the herbicide NOELs based on long-term feeding studies.

Text continued on page 143.

Table 2-45. ADI/dose comparisons for workers on small projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	Above[1]	Above[1]	Above[1]	Above[1]
Picloram	6.3	3.4	2.2	1.1
2,4-D/Picloram	Above/25	Above/13	Above/8.3	Above/4.2
2,4-D/Dicamba	Above/2.5	Above/1.3	Above/Above	Above/Above
Dicamba	Above[1]	Above[1]	Above[1]	Above[1]
Glyphosate	5.6	2.7	1.8	Above[1]
Amitrole	NA[2]	NA[2]	NA[2]	NA[2]
Atrazine	2.1	1.0	Above[1]	Above[1]
Hexazinone	Above[1]	Above[1]	Above[1]	Above[1]
Tebuthiuron	333	167	--[3]	--[3]

---

[1] Worker dose is above the ADI. Consult the NOEL/dose comparisons on the following table.

[2] An ADI has not been set for amitrole. See discussion in Section 2.5 and the NOEL/dose comparisons in the following table.

[3] See discussion in Section 2.4.1.

Table 2-46. NOEL/dose comparisons for workers on small projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	27	14	8.8	4.3
Picloram	1,750	1,100	636	304
Dicamba[1]	139	68	44	22
2,4-D/Picloram	77/7,000	37/3,500	24/2,333	12/1,167
2,4-D/Dicamba[1]	77/500	37/250	24/166	12/89
Glyphosate[2]	555	270	175	86
Amitrole	6.2	3.4	2.3	1.1
Atrazine	208	101	66	32
Hexazinone	3,378	1,689	1,096	539
Tebuthiuron	33,333	16,667	--[3]	--[3]

---

[1] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[2] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

[3] See discussion in Section 2.4.1.

Table 2-47. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with 2,4-D.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	611	6.1	581	5.8
Adolescent resident	607	6.1	577	5.7
Infant resident	459	4.6	437	4.4
Visitor entry	9,090	91	8,333	83
Visitor entry with consumption of sprayed wild food	20	Above[1]	18	Above[1]

[1] Dose is above the ADI.

Table 2-48. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with picloram.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	6,076	22	5,868	21
Adolescent resident	6,071	22	5,868	21
Infant resident	4,818	17	4,657	17
Visitor entry	636,360	2,272	583,330	2,083
Visitor entry with consumption of sprayed wild food	273	Equal[1]	252	Equal[1]

[1] Dose is approximately equal to the ADI.

Table 2-49. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with dicamba.

	Minor mixing error		Major mixing error	
	NOEL[2]	ADI	NOEL[2]	ADI
Adult resident	2,131	8.5	2,058	10
Adolescent resident	2,120	11	2,046	10
Infant resident	1,662	8.3	1,604	8.0
Visitor entry	47,170	236	43,860	219
Visitor entry with consumption of sprayed wild food	96	Above[1]	89	Above[1]

---

[1] Dose is above the ADI.

[2] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

Table 2-50. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with glyphosate.

	Minor mixing error		Major mixing error	
	NOEL[1]	ADI	NOEL[1]	ADI
Adult resident	8,524	85	8,230	82
Adolescent resident	8,480	85	8,183	82
Infant resident	6,648	66	6,414	64
Visitor entry	188,680	1,887	175,440	1,754
Visitor entry with consumption of sprayed wild food	384	3.8	357	3.6

---

[1] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

Table 2-51. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with a 2,4-D/picloram mixture.

	Minor mixing error		Major mixing error	
	NOEL 2,4-D/Picloram	ADI 2,4-D/Picloram	NOEL 2,4-D/Picloram	ADI 2,4-D/Picloram
Adult resident	946/8,526	9.5/30	927/8,424	9.3/30
Adolescent resident	941/8,526	9.4/30	923/8,424	9.3/30
Infant resident	751/7,439	7.5/27	728/6,986	7.3/25
Visitor entry	25,000/777,778	250/2,778	23,810/700,000	238/2,500
Visitor entry with consumption of sprayed wild food	53/1,094	Above[1]/4	48/993	Above[1]/3.5

---

[1] Dose is above the ADI.

Table 2-52. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with 2,4-D/dicamba.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	946/3,026	9.5/15	927/2,990	9.3/15
Adolescent resident	941/3,024	9.4/15	923/2,988	9.2/15
Infant resident	751/2,490	7.5/12	728/2,463	7.3/12
Visitor entry	25,000/192,310	250/960	23,810/178,000	238/890
Visitor entry with consumption of sprayed wild food	53/390	Above[1]/1.9	48/357	Above[1]/1.8

---

[1] Dose is above the ADI.

[2] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

Table 2-53. NOEL/dose[1] comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with amitrole.

	Minor mixing error	Major mixing error
	NOEL	NOEL
Adult resident	22	21
Adolescent resident	22	21
Infant resident	17	16
Visitor entry	2,272	2,083
Visitor entry with consumption of sprayed wild food	Equal[2]	Above[3]

---

[1] As discussed in Section 2.5, an ADI value is not available for amitrole.

[2] Dose is approximately equal to the NOEL.

[3] Dose is above the NOEL.

Table 2-54. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with tebuthiuron.

	NOEL	ADI
Adult resident	2,816	28
Adolescent resident	2,816	28
Infant resident	2,410	24
Visitor entry	35,100,000	351,000

---

[1] Tebuthiuron pellets are not mixed on site. Thus only formulation errors and swath overlap are expected (minor errors).

Table 2-55. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with hexazinone.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	52	10	8,071	10
Adolescent resident	8,278	10	7,981	10
Infant resident	6,419	8.0	6,184	7.7
Visitor entry	90,909	113	83,333	104
Visitor entry with consumption of sprayed wild food	384	Above[1/]	357	Above[1/]

[1/] Dose is above the ADI.

Table 2-56. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a small project sprayed with atrazine.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	52	Above[1/]	52	Above[1/]
Adolescent resident	52	Above[1/]	52	Above[1/]
Infant resident	44	Above[1/]	43	Above[1/]
Visitor entry	70,755	708	65,790	657
Visitor entry with consumption of sprayed wild food	144	1.5	134	1.3

[1/] Dose is above the ADI.

Table 2-57. ADI/dose comparisons for workers on mid-sized projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	Above[1/]	Above[1/]	Above[1/]	Above[1/]
Picloram	5.0	3.1	2.1	1.0
Dicamba	Above[1/]	Above[1/]	Above[1/]	Above[1/]
2,4-D/Picloram	Above[1/]/25	Above[1/]/12	Above[1/]/8.3	Above[1/]/3.6
2,4-D/Dicamba	Above[1/]/2.1	Above[1/]/1.1	Above[1/] Above[1/]	Above[1/] Above[1/]
Glyphosate	5	2.4	1.5	Above[1/]
Amitrole	NA[2/]	NA[2/]	NA[2/]	NA[2/]
Atrazine	1.9	Above[1/]	Above[1/]	Above[1/]
Hexazinone	Above[1/]	Above[1/]	Above[1/]	Above[1/]
Tebuthiuron	286	154	--[3/]	--[3/]

[1/] Dose is above the ADI. Consult the following table for NOEL/dose comparison.

[2/] An ADI has not been set for amitrole. See discussion in Section 2.5.

[3/] See discussion in Section 2.4.1.

Table 2-58. NOEL/dose comparisons for workers on mid-sized projects.

	Recommended Protection Average Dose	Recommended Protection High Dose	Low Protection Average Dose	Low Protection High Dose
2,4-D	24	12	7.8	3.8
Picloram	1,400	875	583	269
Dicamba[1/]	125	60	38	19
2,4-D/Picloram	67/7,000	32/3,500	22/2,333	11/1,006
2,4-D/Dicamba[1/]	67/416	32/227	22/147	11/69
Glyphosate[2/]	500	238	154	76
Amitrole	5.0	3.1	2.1	1.0
Atrazine	188	89	58	29
Hexazinone	238	119	78	38
Tebuthiuron	28,600	15,400	--[3/]	--[3/]

[1/] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[2/] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

[3/] See discussion in Section 2.4.1.

Table 2-59. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with 2,4-D.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	431	Above[1]	408	4.1
Adolescent resident	428	4.3	403	4.0
Infant resident	319	3.2	300	3.0
Visitor entry	1,408	14	1,282	13
Visitor entry with consumption of sprayed wild food	19.6	Above1/	17.9	Above1/

1/ Dose is above the ADI.

Table 2-60. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with picloram.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	4,717	16	4,534	16
Adolescent resident	4,714	16	4,531	16
Infant resident	3,686	13	3,518	13
Visitor entry	100,000	357	87,500	313
Visitor entry with consumption of sprayed wild food	269	Equal1/	250	Equal1/

1/ Dose is approximately equal to the ADI.

Table 2-61. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with dicamba.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	1,645	8.2	1,582	7.9
Adolescent resident	1,634	8.1	1,572	7.9
Infant resident	1,262	6.3	1,200	6.0
Visitor entry	7,143	35	6,410	32
Visitor entry with consumption of sprayed wild food	96	Above	89	Above

---

[1] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

Table 2-62. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with glyphosate.

	Minor mixing error		Major mixing error	
	NOEL[1/]	ADI	NOEL[1/]	ADI
Adult resident	6,580	66	6,328	43
Adolescent resident	6,536	65	6,288	63
Infant resident	5,048	50	4,800	48
Visitor entry	28,570	286	25,640	256
Visitor entry with consumption of sprayed wild food	384	3.9	356	3.6

[1/] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

Table 2-63. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with 2,4-D/picloram.

	Minor mixing error		Major mixing error	
	NOEL 2,4-D/Picloram	ADI 2,4-D/Picloram	NOEL 2,4-D/Picloram	ADI 2,4-D/Picloram
Adult resident	757/7,769	7.6/28	735/7,600	7.4/27
Adolescent resident	752/7,769	7.5/28	730/7,600	7.3/27
Infant resident	588/6,352	5.9/23	568/6,240	5.7/22
Visitor entry	3,700/350,000	370/1,250	3,570/350,000	357/1,250
Visitor entry with consumption of sprayed wild food	52/1,092	Above[1]/3.9	48/1,000	Above[1]/3.6

[1] Dose is above the ADI.

Table 2-64. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with 2,4-D/dicamba.

	Minor mixing error		Major mixing error	
	NOEL[2]	ADI	NOEL[2]	ADI
Adult resident	757/2,750	7.6/14	735/2,690	7.4/13
Adolescent resident	752/2,750	7.5/14	730/2,690	7.3/13
Infant resident	588/2,230	5.9/11	568/2,174	5.7/11
Visitor entry	3,700/27,800	37/138	3,570/27,800	357/138
Visitor entry with consumption of sprayed wild food	52/390	Above[1]/2.0	48/360	Above[1]/1.8

---

[1] Doses are above the ADI.

[2] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

Table 2-65. NOEL/dose[1] comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with amitrole.

	Minor mixing error	Major mixing error
	NOEL[2]	NOEL[2]
Adult resident	16	16
Adolescent resident	16	16
Infant resident	13	13
Visitor entry	357	313
Visitor entry with consumption of sprayed wild food	1.0	Above[2]

---

[1] An ADI is not available for amitrole.

[2] Dose is above the NOEL.

Table 2-66. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with hexazinone.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	6,410	8.0	6,173	7.7
Adolescent resident	6,329	7.9	6,060	7.6
Infant resident	4,830	6.0	4,587	5.7
Visitor entry	14,085	18	12,821	16
Visitor entry with consumption of sprayed wild food	384	Above[1]	357	Above[1]

---

[1] Dose is above the ADI.

Table 2-67. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-sized project sprayed with atrazine.

	Minor mixing error		Major mixing error	
	NOEL	ADI	NOEL	ADI
Adult resident	52	Above	52	Above
Adolescent resident	52	Above	52	Above
Infant resident	45	Above	44	Above
Visitor entry	10,714	107	9,600	96
Visitor entry with consumption of sprayed wild food	146	1.5	135	1.4

Table 2-68. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a mid-size project sprayed with tebuthiuron.

	Minor mixing error[1/]	
	NOEL	ADI
Adult resident	2,816	28
Adolescent resident	2,816	28
Infant resident	2,410	24
Visitor entry	5,128,000	51,280

[1/] Tebuthiuron pellets are not mixed on site. Thus, only formulation errors and swath overlap are expected (minor errors).

Table 2-69. ADI/dose comparisons for backpack workers on large projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	Above[1]	Above[1]	Above[1]	Above[1]
Picloram	5.0	3.1	2.1	1.0
Dicamba	Above[1]	Above[1]	Above[1]	Above[1]
2,4-D/Picloram	Above[1]/25	Above[1]/12	Above[1]/8.3	Above[1]/3.6
2,4-D/Dicamba	Above[1]/2.1	Above[1]/1.1	Above[1]/Above[1]	Above[1]/Above[1]
Glyphosate	5	2.4	1.5	Above[1]
Amitrole	NA[2]	NA[2]	NA[2]	NA[2]
Atrazine	1.9	Above[1]	Above[1]	Above[1]
Hexazinone	Above[1]	Above[1]	Above[1]	Above[1]
Tebuthiuron	286	154	--[3]	--[3]

---

[1] Dose is above the ADI. Consult the following table for NOEL/dose comparison.

[2] An ADI has not been set for amitrole. See discussion in Section 2.5.

[3] See discussion in Section 2.4.1.

Table 2-70. NOEL/dose comparisons for backpack workers on large projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	24	12	7.8	3.8
Picloram	1,400	875	583	269
Dicamba[1]	125	60	38	19
2,4-D/Picloram	67/7,000	32/3,500	22/2,333	11/1,006
2,4-D/Dicamba[1]	67/416	32/227	22/147	11/69
Glyphosate[2]	500	238	154	76
Amitrole	5.0	3.1	2.1	1.0
Atrazine	188	89	58	29
Hexazinone	238	119	78	38
Tebuthiuron	28,606	15,400	--[3]	--[3]

---

[1] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[2] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

[3] See discussion in Section 2.4.1.

Table 2-71. NOEL/dose and ADI/dose comparisons for supervisors on large projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	909	9.1	208	2.1
Picloram	70,000	250	14,000	50
Dicamba[2/]	5,000	50	1,042	10
2,4-D/Picloram	2,500/233,333	25/292	556/70,000	5.6/88
2,4-D/Dicamba[2/]	2,500/25,000	25/125	556/4,166	5.6/42
Glyphosate[3/]	20,000	200	4,167	42
Amitrole	250	NA[1/]	50	NA[1/]
Atrazine	5,357	54	1,562	15
Tebuthiuron	4,000,000	40,000	800,000	8,000
Hexazinone	9,090	11	2,083	2.6

[1/] An ADI for amitrole is not available.

[2/] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the value listed here.

[3/] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the value listed here.

Table 2-72. NOEL/dose and ADI/dose comparisons for truck drivers on large projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	33	Above[1]	8.8	Above[1]
Picloram	2,333	8.3	636	2.3
Dicamba[2]	166	Above[1]	44	Above[1]
2,4-D/Picloram	91/10,000	Above[1]/36	23/2,333	Above[1]/8.3
2,4-D/Dicamba[2]	91/625	Above[1]/3.1	23/192	Above[1]/1.0
Glyphosate[3]	666	6.7	178	1.8
Amitrole	8.3	Above[1]	2.3	Above[1]
Atrazine	250	2.5	67	Above[1]
Tebuthiuron	134,000	1,300	35,000	350
Hexazinone	333	Above[1]	88	Above[1]

---

[1] Dose is above the ADI.

[2] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[3] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

Table 2-73. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a large project sprayed with 2,4-D or picloram.

	2,4-D		Picloram	
	NOEL	ADI	NOEL	ADI
Adult resident	179	1.8	2,244	8.0
Adolescent resident	177	1.8	2,244	8.0
Infant resident	115	1.1	2,154	7.6
Visitor entry	909	9.1	70,000	250
Visitor entry with consumption of sprayed wild food	19.5	Above[1/]	19.2	Above[1/]

[1/] Dose is above the ADI.

Table 2-74. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a large project sprayed with glyphosate or dicamba.

	Dicamba		Glyphosate	
	NOEL	ADI	NOEL	ADI
Adult resident	781	3.9	3,125	31
Adolescent resident	774	3.9	3,096	31
Infant resident	527	2.6	2,110	21
Visitor entry	5,000	25	20,000	200
Visitor entry with consumption of sprayed wild food	96	Above[1/]	384	3.8

[1/] Dose is above the ADI.

Table 2-75. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a large project sprayed with a 2,4-D/picloram mixture.

	2,4-D		Picloram	
	NOEL	ADI	NOEL	ADI
Adult resident	388	3.9	5,335	19
Adolescent resident	384	3.8	5,334	19
Infant resident	263	2.6	4,035	14
Visitor entry	2,500	25	233,333	833
Visitor entry with consumption of sprayed wild food	50	Above[1]	1,167	4.1

[1] Dose is above the ADI level.

Table 2-76. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a large project sprayed with a 2,4-D/dicamba mixture.

	2,4-D		Dicamba	
	NOEL	ADI	NOEL	ADI
Adult resident	388	3.9	1,880	9.4
Adolescent resident	384	3.8	1,866	9.3
Infant resident	263	2.6	1,404	7.0
Visitor entry	2,500	25	5,000	25
Visitor entry with consumption of sprayed wild food	50	Above[1]	417	2.1

[1] Dose is above the ADI level.

Table 2-77. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a large project sprayed with amitrole or hexazinone.

	Amitrole		Hexazinone	
	NOEL	ADI[1]	NOEL	ADI[1]
Adult resident	8.0	---	3,040	3.8
Adolescent resident	8.0	---	2,994	3.7
Infant resident	5.4	---	2,016	2.5
Visitor entry	23	---	9,090	11
Visitor entry with consumption of sprayed wild food	Above[2]	---	389	Above[2]

---

[1] An ADI is not available for amitrole.

[2] Dose is above the NOEL.

Table 2-78. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a large project sprayed with atrazine or tebuthiuron.

	Atrazine		Tebuthiuron	
	NOEL	ADI	NOEL	ADI
Adult resident	51	Above[1]	2,816	28
Adolescent resident	51	Above[1]	2,816	28
Infant resident	44	Above[1]	2,410	24
Visitor entry	7,500	75	5,128,000	51,280
Visitor entry with consumption of sprayed wild food	144	1.4	---	---

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[1] Dose is above the ADI.

Table 2-79. NOEL/dose and ADI/dose comparisons for truck drivers on right-of-way and riparian projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	50	Above[1]	12	Above[1]
Picloram	3,500	12	875	Above[1]
Dicamba[2]	250	1.2	62	Above[1]
2,4-D/Picloram	188/1,400	1.9/50	33/3,500	Above[1]/12
2,4-D/Dicamba[2]	188/833	1.9/4.2	33/250	Above[1]/1.2
Glyphosate[3]	1,000	10	250	2.5
Amitrole	2.5	--[4]	Above[1]	--[4]
Atrazine	375	3.8	94	Above[1]
Hexazinone	500	Above[1]	125	Above[1]
Tebuthiuron	200,000	2,000	50,000	500

---

[1] Worker dose is above the ADI.

[2] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[3] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

[4] An ADI for amitrole is not available.

Table 2-80. ADI/dose comparisons for spot sprayers (backpack) on right-of-way and riparian projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	Above[1]	Above[1]	Above[1]	Above[1]
Picloram	5	2.5	1.7	Above[1]
Dicamba	Above[1]	Above[1]	Above[1]	Above[1]
2,4-D/Picloram	Above[1]/25	Above[1]/12	Above[1]/6.2	Above[1]/3.6
2,4-D/Dicamba	Above[1]/1.8	Above[1]/1.0	Above[1]	Above[1]
Glyphosate	4.0	2.0	1.3	Above[1]
Amitrole	NA[2]	NA[2]	NA[2]	NA[2]
Atrazine	1.5	Above[1]	Above[1]	Above[1]
Hexazinone	Above[1]	Above[1]	Above[1]	Above[1]
Tebuthiuron	800	400	257	129

---

[1] Worker dose is above the ADI.

[2] An ADI for amitrole is not available.

Table 2-81. NOEL/dose comparisons for spot sprayers (backpack) on right-of-way and riparian projects.

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	20	10	6.6	3.2
Picloram	1,400	700	466	226
Dicamba[2/]	100	51	32	16
2,4-D/Picloram	58/7,000	27/3,500	17/1,750	8.9/1,000
2,4-D/Dicamba[2/]	58/357	27/208	17/131	8.9/68
Glyphosate[3/]	400	204	131	65
Amitrole	5.0	2.5	1.7	Above[1/]
Atrazine	150	77	49	24
Hexazinone	204	101	65	32
Tebuthiuron	80,000	40,000	25,700	12,900

[1/] Worker dose is above the NOEL.

[2/] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[3/] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

Table 2-82. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of right-of-way and riparian projects sprayed with 2,4-D or picloram.

	2,4-D		Picloram	
	NOEL	ADI	NOEL	ADI
Adult resident	158	1.6	2,220	7.9
Adolescent resident (dermal and oral dose)	95	Above[1/]	1,652	5.9
Adolescent resident (oral doses only)	124	1.2	1,731	6.2
Infant resident	108	1.1	1,531	5.4
Visitor entry	1,786	18	125,000	446

[1/] Dose is above the ADI.

Table 2-83. NOEL/dose and ADI/dose comparisons for maximum-exposed residents in the vicinity of right-of-way and riparian projects sprayed with dicamba, tebuthiuron, or glyphosate.

	Dicamba[1]		Glyphosate[2]		Tebuthiuron	
	NOEL	ADI	NOEL	ADI	NOEL	ADI
Adult resident	789	3.9	3,155	32	6,000	60
Adolescent resident (dermal and oral dose)	481	2.4	1,900	19	NA[3]	NA[3]
Adolescent resident (oral doses only)	618	3.1	2,472	25	4,740	47
Infant resident	547	2.7	2,187	22	4,237	42
Visitor entry	8,929	44	35,700	357	6,667,000	66,670

---

[1] Table entries for dicamba are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about 10 times the values listed here.

[2] Table entries for glyphosate are based on the reproductive effects NOEL. NOEL/dose comparisons using the NOEL for general toxic effects would be about three times the values listed here.

[3] Tebuthiuron is applied in pellet form only. Therefore, drift and dislodgable residues on vegetation cannot contact the general public.

Table 2-84. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of right-of-way and riparian projects sprayed with a 2,4-D/picloram mixture.

	2,4-D/Picloram	
	NOEL	ADI
Adult resident	420/8,776	4.2/31
Adolescent resident (dermal and oral dose)	254/6,517	2.5/23
Adolescent resident (oral doses only)	330/6,654	3.3/24
Infant resident	288/6,134	2.9/22
Visitor entry	4,762/700,000	48/2,500

Table 2-85. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of right-of-way and riparian projects sprayed with a 2,4-D/dicamba mixture.

	2,4-D/Dicamba	
	NOEL	ADI
Adult resident	420/3,145	4.2/15
Adolescent resident (dermal and oral dose)	254/1,900	2.5/9.5
Adolescent resident (oral dose only)	330/2,466	3.3/12
Infant resident	288/2,172	2.9/11
Visitor entry	4,762/35,000	48/175

Table 2-86. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of right-of-way and riparian projects sprayed with amitrole, atrazine, or hexazinone.

	Amitrole		Atrazine		Hexazinone	
	NOEL	ADI	NOEL	ADI	NOEL	ADI
Adult resident	7.9	NA[1]	1,110	11	2,960	3.7
Adolescent resident (dermal and oral doses)	5.9	NA[1]	696	7.0	1,506	1.9
Adolescent resident (oral doses)	6.2	NA[1]	886	8.9	2,364	3.0
Infant resident	5.4	NA[1]	775	7.7	2,050	2.6
Visitor entry	446	NA[1]	13,393	134	17,857	22

---

[1] An ADI for amitrole is not available.

Table 2-87. NOEL/dose and ADI/dose comparisons for pilots on aerial spray projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	59	Above[1]	14	Above[1]
2,4-D/Dicamba	167/1,250	1.7/12	40/277	Above[1]/1.4
Picloram	3,500	12	1,000	3.6
Tebuthuiron	200,000	2,000	66,667	667

---

[1] Dose is above the ADI.

Table 2-88. NOEL/dose and ADI/dose comparisons for mixer/loaders on aerial spray projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	53	Above[1/]	19	Above[1/]
2,4-D/Dicamba	142/833	1.4/4.2	53/357	Above[1/]/1.8
Picloram	3,500	12	1,400	5.0
Tebuthiuron	200,000	2,000	66,667	667

[1/] Dose is above ADI.

Table 2-89. NOEL/dose and ADI/dose comparisons for supervisors on aerial spray projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	333	3.3	100	1.0
2,4-D/Dicamba	1,111	11	250	2.5
Picloram	23,333	83	5,833	58
Tebuthiuron	666,000	6,660	167,000	1,670

Table 2-90. NOEL/dose and ADI/dose comparisons for observers on aerial spray projects.

	Average Dose		High Dose	
	NOEL	ADI	NOEL	ADI
2,4-D	2,000	20	714	7.1
2,4-D/Dicamba	5,000/35,000	50/178	2,000/12,500	20/62
Picloram	140,000	500	50,000	178
Tebuthiuron	4,000,000	40,000	2,000,000	20,000

Table 2-91. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a project aerially sprayed with 2,4-D.

	Worst Case		Routine	
	NOEL	ADI	NOEL	ADI
Adult resident with water consumption	24	Above[1/]	715	7.2
Adult resident without water consumption	133	1.3	1,395	14
Adolescent resident with water consumption	24	Above[1/]	760	7.6
Adolescent resident without water consumption	132	1.2	1,395	14
Infant resident with water consumption	16	Above[1/]	543	5.4
Infant resident without water consumption	99	1.0	1,190	11
Visitor entry	100	1.0	333	3.3
Visitor entry with consumption of sprayed wild food	16	Above[1/]	19	Above[1/]

[1/] Dose is above the ADI.

Table 2-92. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a project aerially sprayed with picloram.

	Worst Case		Routine	
	NOEL	ADI	NOEL	ADI
Adult resident with water consumption	333	1.2	6,645	24
Adult resident without water consumption	1,740	6.2	9,813	35
Adolescent resident with water consumption	367	1.3	6,910	24
Adolescent resident without water consumption	1,740	1.2	9,813	35
Infant resident with water consumption	226	Above[1]	5,243	19
Infant resident without water consumption	1,176	4.1	8,380	30
Visitor entry	5,830	21	23,300	83
Visitor entry with consumption of sprayed wild food	257	Above[1]	266	Above[1]

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[1] Dose above the ADI.

Table 2-93. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a project aerially treated with tebuthiuron.

	Worst Case		Routine	
	NOEL	ADI	NOEL	ADI
Adult resident with water consumption	830	8.3	NA[1]	NA[1]
Adult resident without water consumption	2,817	28	2,817	28
Adolescent resident with water consumption	904	9.0	NA[1]	NA[1]
Adolescent resident without water consumption	2,817	28	2,817	29
Infant resident with water consumption	600	6	NA[1]	NA[1]
Infant resident without water consumption	2,410	24	2,410	24
Visitor entry	167,000	1,670	667,000	6,670

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[1] Drift of tebuthiuron pellets and consequent aquatic impacts are not expected under routine conditions.

Table 2-94. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a project aerially sprayed with 2,4-D/dicamba (worst-case conditions).

	2,4-D		Dicamba	
	NOEL	ADI	NOEL	ADI
Adult resident with water consumption	67	Above[1/]	434	2.2
Adult resident without water consumption	334	3.3	1,600	8.0
Adolescent resident with water consumption	76	Above[1/]	466	2.3
Adolescent resident without water consumption	333	3.3	1,600	8.0
Infant resident with water consumption	39	Above[1/]	280	1.5
Infant resident without water consumption	227	2.3	1,140	5.7
Visitor entry	250	2.5	1,250	6.2
Visitor entry with consumption of sprayed wild food	42	Above[1/]	416	2.1

[1/] Dose is above the ADI.

Table 2-95. NOEL/dose and ADI/dose comparisons for maximum-exposed residents and visitors in the vicinity of a project aerially sprayed with 2,4-D/dicamba (routine conditions).

	2,4-D		Dicamba	
	NOEL	ADI	NOEL	ADI
Adult resident with water consumption	1,050	10	3,145	16
Adult resident without water consumption	1,400	14	3,517	18
Adolescent resident with water consumption	1,100	11	3,160	16
Adolescent resident without water consumption	1,400	14	3,517	18
Infant resident with water consumption	850	8.5	2,616	13
Infant resident without water consumption	1,200	12	3,000	15
Visitor entry	1,110	11	6,250	31
Visitor entry with consumption of sprayed wild food	48	Above[1]	416	2.1

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[1] Dose is above the ADI.

The maximum estimated dose to an adolescent who spends the day in the vicinity of a right-of-way spray project could exceed slightly the ADI for the herbicide 2,4-D. This dose would be about 95 times lower than the NOEL based on long-term dosing studies. Since the adolescent dose would occur at most only once at the levels predicted, health effects would be unlikely.

Doses to members of the general population from atrazine-treated, open-range projects could exceed the ADI for atrazine if the person consumes a large amount of beef that grazed exclusively on atrazine-treated range. Label restrictions prohibit grazing cattle on atrazine-treated range for 3 to 7 months after treatment depending on application timing. The Forest Service would enforce these label restrictions to prevent this exposure. In addition, the small amount of atrazine applied in a year on scattered sites ensures that doses on the order estimated in the analysis are virtually impossible.

Under routine conditions, the aerial application of herbicides would not significantly increase public exposure over that received during ground application. However, misapplications and accidents during aerial application could increase public exposure. For example, if a plane directly sprayed a small stream with 2,4-D and an adult immediately drank a liter of water from this stream, his dose would exceed the ADI for 2,4-D. Generally, the doses from exposure to all other aerially applied herbicides (picloram, dicamba, and tebuthiuron) are below the ADI's except in the case of a direct spray or spill of herbicides over bystanders (see Section 3.0).

Although most doses to the general public are well below NOEL and ADI levels based on animal tests, it is possible that a small percentage of the human population may be very sensitive to chemical exposures. For example, the medical literature has reported several cases of peripheral neuropathy resulting from exposure to 2,4-D (see Goldstein et al. 1959, Berkley and Magee 1963, and Berwick 1970). Peripheral neuropathy is the disruption of the nervous system characterized by some or all of the following symptoms: numbness in hands and feet, loss of balance, aching in extremities, fatigue, and nausea. Recovery in some cases is very prolonged and may not be complete even after several years (Goldstein et al. 1959).

Although many of the reported cases of peripheral neuropathy occurred after massive doses, effects in some people have been noted at much lower apparent doses. The conclusions of Berkley and Magee (1963) seem appropriate.

> "Despite the extensive use of 2,4-D preparations, resultant peripheral neuropathy is very rare, and an affected individual probably has some predisposition to neuropathy or susceptibility to the toxin. Nevertheless, as it cannot be determined who is predisposed or susceptible, and as no antidote to 2,4-D intoxication is known, prevention is simpler than treatment."

It is also possible that idiosyncratic toxic responses as yet undetected by the medical community could result from the exposure to other herbicides or combinations of herbicides. Once again, prevention of exposure for both workers and the general public is the most prudent course.

2.6.2 Discussion of ADI and NOEL Comparisons for Worker Doses

In general, of the various populations exposed to pesticides, the workers applying herbicides incur the highest risk of health impacts. In particular, backpack applicators are likely to receive the highest doses. This section will summarize the trends in worker doses for each herbicide.

2.6.2.1 Amitrole

Amitrole application could pose a risk of thyroid effects based on a comparison of worker doses with dose levels that disrupt thyroid functions in animals. Although an ADI for amitrole has not been set by EPA because it is not approved for use in agricultural commodities, the proximity of most doses to the amitrole NOEL level is cause for concern. Workers applying amitrole with careless techniques and little protective clothing for relatively short periods of time (perhaps as little as 30 days) stand a significant risk of disrupting thyroid function.

Even with allowances for protective clothing, the average worker dose is below the NOEL, based on animal tests, by only a factor of five. Since humans could be at least 6 to 12 times more sensitive on a body weight basis than test animals, this dose could also impact worker health.

Measures that could reduce human health impacts to workers include use of rubber gloves and rubber boots, careful application techniques, and personal hygiene to avoid dermal and oral exposure. Limitations on worker exposure to amitrole to less than 5 days per year would also reduce the probability of cumulative impacts on thyroid function although data are not available to indicate whether the risk could be eliminated.

Worker dose under all protective scenarios are at least 100 times less than dose levels that cause fetotoxic effects.

2.6.2.2 Atrazine

Assuming good protective techniques and careful application habits, worker doses from atrazine applications can be at or below the ADI for atrazine. Conversely, careless techniques could result in doses that are as little as 25 times lower than the NOEL. Worker doses, under all protection scenarios are at least 100 times less than dose levels causing fetotoxic effects in humans.

2.6.2.3 2,4-D

All worker dose estimates for backpack workers, truck applicators, pilots, and mixer/loaders are above the ADI for 2,4-D. Backpack worker doses range from as little as one-third the NOEL levels to 24 times less than the NOEL. At dose levels above the NOEL, changes in kidney function in test animals were observed with as little as 90 days' dosing. Tests on the reversibility of the changes in kidney function have not been conducted. Backpack workers are at some risk of effects on kidney function. Application techniques and the use of rubber gloves, rubber boots, and long-sleeved shirts can reduce this impact.

The dose estimates for truck applicators, pilots, and mixer/loaders are lower than backpack worker dose estimates, thus the risk of kidney effects is lessened although not completely eliminated. For all worker functions there exists the possibility of neuropathological effects as described above in the general population section.

Worker doses (backpack workers, truck applicators, pilots, and mixer/loaders) range from 80 times less than the fetotoxic NOEL under low protection scenarios, to 675 times less under recommended protection scenarios.

2.6.2.4 Dicamba

Of the herbicides analyzed here, dicamba poses the greatest risk of fetotoxic or reproductive effects for pregnant applicators. The NOEL of 2.5 mg/kg/day was based on observed disruption of reproductive functions in female rats. Dose estimates for backpack workers, truck applicators, pilots, and mixer/loaders range from 16 to 1,250 times less than this NOEL.

A NOEL of 25 mg/kg/day has been established for general systemic toxic effects (kidney and liver function) based on a 90-day feeding study. Thus, the risk of adverse health effects would be much lower for male applicators. NOEL/dose comparisons would range from 160 to 12,500.

2.6.2.5 Glyphosate

Except for the highest dose estimate for backpack workers under the low protection scenario, all worker dose estimates are below the ADI for glyphosate.

2.6.2.6 Hexazinone

NOEL/dose comparison factors for hexazinone application under recommended protection scenarios are all at least 100. Under low protection scenarios, the high doses could be as little as 32 times less than the NOEL.

The fetotoxicity NOEL for hexazinone is 50 mg/kg. This NOEL is at about 200 times higher than the highest worker dose estimate for any project type and protection scenario.

2.6.2.7 Picloram

Picloram is similar to glyphosate in that all worker dose estimates are below the ADI except the highest dose estimate under the low protection backpack worker scenario. The NOEL for general systemic effects is about 226 times higher than this high-dose estimate.

2.6.2.8 Tebuthiruon

Because tebuthiuron is applied in pellet form, the worker exposure to drift and herbicide-treated foliage is minimized. All worker dose estimates are below the ADI for tebuthiruon.

2.7 CARCINOGENIC AND MUTAGENIC EFFECTS OF HERBICIDES

The fear of cancer and other mutations from pesticide exposure is a salient response of many people. For this reason and because of unique considerations in the mechanism of mutagenesis, carcinogenic and heritable mutagenic effects are discussed separately from other chronic effects.

For our purposes, a mutagen is a chemical substance or mixture of substances that can induce alterations in the DNA of either somatic or germinal cells (U.S. EPA 1984a). Somatic ells refer to the body's general cells contained in organs such as the liver; germinal cells are contained in the reproductive organs, i.e, the gonads, and contain the genetic basis for new life. Mutations carried in germ cells are inherited by future generations and may contribute to genetic disease, whereas mutations occurring in somatic cells have been implicated in the development of several disease states, including cancer (U.S. EPA 1984a). Although widely assumed that tumor formation arises from mutation, other non-mutagenic causes of heritable changes in somatic cells are possible (see discussion in OSTP 1985). This analysis assumes, however, that mutagenesis plays a major role in the development of many cancers.

When evaluating the ability of the pesticides to produce genotoxic effects such as tumor initiation (cancer) or heritable mutations (birth defects), this analysis does not use NOELs or threshold doses. Thresholds are not assumed because it is conceivable that only one or a few molecules of an active chemical may cause changes in DNA that could form neoplastically transformed cells (tumors) or heritable mutagenic effects.

Individual and population risks of developing cancer can be quantified using various models if there is scientific evidence to suggest a chemical is a carcinogen. Since quantitative risk models are not available for estimating the possibility of transmitting mutations across generations (heritable mutations), a multistep process of evaluating a pesticide's ability to cause mutations in germinal cells assesses qualitatively the mutagenic risk in humans (U.S. EPA 1984a).

The first step in the assessment of mutagenic risk involves an analysis of the evidence of a pesticide's ability to cause mutations in bacteria, microorganisms, insects, plants, mammalian cells in culture and germinal cells in whole animals. The second step involves an analysis of its ability to produce these effects in mammalian gonads. Greater weight is placed on tests that show changes in germinal tissues than in somatic cells, on tests performed in vivo (within the body) rather than in vitro (outside the body), and in mammalian species rather than in submammalian species (U.S. EPA 1984a). Table 2-96, provided by Dr. David Brusick with Litton Bionetics, Inc., presents a listing of various tests and their value in predicting a chemical's mammalian carcinogenic and heritable mutagenic potential.

Extensive reviews of the mutagenesis literature for the herbicides of interest are provided in Agriculture Handbook No. 633 (USDA, Forest Service 1984 and

1986a). Highlights of these reviews and EPA mutagenesis data summaries provided in tolerance determinations are provided below. Unless otherwise indicated, all test results are summarized from Agriculture Handbook 633 (USDA, Forest Service 1984 and 1986a). In reviewing these results, the trends are most significant since no individual mutagenesis test is perfectly predictive and every test can give false positives and false negatives.

2.7.1 Mutagenic Potential of Herbicides

2.7.1.1 Amitrole Mutagenesis Tests

Amitrole tested negative (no mutation) in 49 tests with various strains of *Salmonella typhimurium* in Ames mutagenicity assays. Amitrole was negative in tests with the Chinese hamster ovary (CHO test is indicative of heritable mutagenic potential). Amitrole was nonmutagenic (negative results) in tests with human lymphocytes and various mouse cellular systems.

Amitrole tested positive when treated with equivalent amounts of nitrite, indicating that amitrole can be nitrosated to a mutagenic compound. Similar tests with a metabolically activated amitrole also gave positive results for mutagenesis. Amitrole also appears to damage DNA as evidenced by positive responses for unscheduled DNA synthesis observed in human cells.

Table 2-96. A summary of the possible roles for selected short-term tests in chemical hazard assessment.

General assay type	Identifies carcinogenic potential	Identifies inheritable mutagenic potential
Microbial Assays		
Ames Reverse Mutation Test	++	+
Reverse Mutation in E. coli WP2 and Related Strains	+	+
Bacterial DNA Repair Tests	+	NA
Yeast Mutation Tests	+	++
Yeast Mitotic Recombination	+	NA
In Vitro Mammalian Cell Assays		
Mouse Lymphoma Assay (TK)	+	++
CHO or V79 Mutation Assays (HGPRT)	+	++
Unscheduled DNA Synthesis (UDS)	++	NA
Chromosome Aberrations	+	++
Sister Chromatid Exchange (SCE)	++	NA
Cell Transformation	++	NA
In Vivo Mammalian Assays		
SCE	+	NA
Dominant Lethal Assay	NA	++
Cytogenetic Analysis (aberrations)	+	++
Micronucleus Assay	+	+
Spermhead Abnormality Assay	NA	(+)
Heritable Translocation Assay in Mice	NA	+
Specific Locus Assay in Mice	NA	++
DNA Adduct Formation	+	(+)
UDS Assays	+	(+)
In Vivo Submammalian Assays		
Drosphila Assays	+	++
Plant Cytogenetics	NA	(+)

+ = Applicable
++ = Greater applicability for this role
NA = Not applicable
(+) = Possible application under limited conditions

(Source: Dr. David J. Brusick, Litton Bionetics, Inc.)

Test evidence does not indicate that amitrole can cause heritable mutations. However, as discussed below in Section 2.7.9, this analysis assumes that amitrole is a carcinogen.

2.7.1.2 Atrazine Mutagenesis Tests

A summary and review of mutagenesis tests with atrazine reveal both positive and negative results (USDA, Forest Service 1984). Although a large number of tests are negative, many of the tests with metabolically activated atrazine proved positive in tests indicative of carcinogenic potential. As discussed below in Section 2.7.9, this analysis assumes that atrazine is a carcinogen.

Three positive responses in mutagenesis studies were in tests with the fruit fly that measured gene mutations in germ cells. Positive results were also obtained in tests with mice which measured chromosome alterations in germ cells. Positive responses in these assays indicate a potential for mutagenic hazard. Chromosome aberrations in bone marrow cells in vivo support this conclusion. However, these in vivo responses were observed only at very high levels of atrazine equal to or exceeding 1,500 mg/kg. Although these results show that atrazine must be viewed as mutagenic at high levels of exposure, the risk to humans at low doses would be minimal.

2.7.1.3 2,4-D Mutagenesis Tests

As reviewed in Agriculture Handbook No. 633 (USDA, Forest Service 1984), 2,4-D was nonmutagenic in most of the microbial systems investigated. In tests with human lymphocytes, both positive and negative results were reported. Assays for detecting unscheduled DNA synthesis with human embryonic lung cells both in the presence and absence of metabolic activation systems were negative. However, as is discussed below, 2,4-D will be assumed to be a carcinogen based on ambiguous evidence from whole animal tests and some evidence of carcinogenic association in human epidemiological studies.

Tests designed to reveal potential for initiation of heritable mutations including tests with Drosophila and tests for mouse dominant lethal mutations are all reported to give nonmutagenic results. However, EPA has requested additional data on the mutagenicity of 2,4-D. In the interim, the worst case assumes that 2,4-D is a mutagen.

2.7.1.4 Dicamba Mutagenesis Tests

Dicamba has not shown mutagenic potential in mutagenesis tests ranging from *S. typhimurium* to human fibroblast cells. Dicamba has been negative in all of the test systems reported in Agriculture Handbook 633 except those measuring relative toxicity. Based on these results, dicamba is not considered mutagenic.

2.7.1.5 Glyphosate Mutagenesis Tests

As reported in Agriculture Handbook No. 633 (USDA, Forest Service 1984), microbial mutagenesis tests with eight strains of bacteria and yeasts all showed no mutagenic effects for glyphosate. No evidence of mutagenicity was observed in the dominant lethal mutation assays with mice.

2.7.1.6 Hexazinone Mutagenesis Tests

As reported in Agricultural Handbook No. 633 (USDA, Forest Service 1984), hexazinone gave negative results for mutagenesis in a variety of tests designed to show carcinogenic potential. In test systems with Chinese hamster ovary (CHO) cells, mutagenic results were reported in a subset of in vitro cytogenic assays. However, nonmutagenic results were observed in a CHO cell point mutation assay. Likewise, hexazinone caused no chromosomal aberations in an *in vivo* bone marrow cytogenic assay using Sprague Dawley rats. Based on the results, hexazinone is unlikely to cause heritable mutations.

2.7.1.7 Picloram Mutagenesis Tests

Picloram has shown no mutagenic potential in a standard battery of microbial mutagenesis assays. Only in unvalidated assay systems did picloram show mutagenic activity (USDA, Forest Service 1984).

In a study to determine possible cytogenic effects on bone marrow cells in animals, picloram was fed to rats at dosages up to 2,000 mg/kg without adverse effects. EPA has requested additional picloram mutagenicity studies. The worse-case assumption is that picloram is a mutagen.

As is discussed below, ambiguous evidence from whole animal carcinogenesis studies form the basis for assuming that picloram is a carcinogen.

2.7.1.8 Tebuthiuron Mutagenesis Tests

Mutagenicity tests of tebuthiuron were negative in five of six systems used. Tests involved several strains of *Salmonella typhimurium* and *Escherichia coli* as well as whole animal and in vitro tests with cells of rats, mice, and hamsters (Todd et al. 1974). Tests for heritable mutations all indicated that tebuthiuron was nonmutagenic.

The sixth study was conducted both with and without metabolic activation. Tebuthiuron was not mutagenic in tests with metabolic activation. When cells were cultured without metabolic activation, a two- to threefold increase in mutation frequency occurred when tebuthiuron exceeded 400 ug/ml of culture solution (Todd et al. 1974). The number of negative tests and the dose levels required to induce a mutagenic effect indicate that the mutagenic potential of tebuthiuron is very low.

2.7.2 Carcinogenic Potential of Herbicides

2.7.2.1 2,4-D Cancer Studies

In keeping with the conservative basis of these risk analyses, a herbicide is considered to have carcinogenic potential if whole animal test data indicates oncogenic activity, no matter how weak.

The U.S. EPA is currently reviewing toxicity test data for 2,4-D and has requested additional testing of this compound. Two chronic toxicity feeding studies (2-year) were completed in 1986. A feeding study with $B_6C_3F_1$ mice showed no difference in tumor formation in male or female mice fed 2,4-D at 0, 1, 15, or 45 mg/kg body weight (Machotka 1986). A 2-year feeding with F344 rats showed a slight increase in brain tumor formation in the highest feed level. In male rats fed 2,4-D at 0, 1, 5, 15, and 45 mg/kg body weight (60 rats at each level) the brain tumors were seen in 1, 0, 0, 2, and 5 rats, respectively. Female rats showed no increase in brain tumors.

A review of the rat study by Adalbert Koestner, Chairman of the Department of Pathology, Michigan State University, examines whether this increase in brain tumors was due to 2,4-D dose or was a chance occurrence. For a variety of reasons outlined in his review (Koestner undated), Koestner believes the increase was due to natural biological variability. Nonetheless, this analysis will treat these findings as possible carcinogenic activity from 2,4-D doses.

In addition to these recent studies, two other studies of 2,4-D carcinogenic activity are also available. As part of a study involving a large number of chemicals, Innes et al. (1969, as seen in USDA, Forest Service 1984) exposed mice of two strains orally to two different formulations of 2,4-D for 18 months. Eighteen mice of each sex and each strain were exposed to each formulation. Exposure to 2,4-D did not result in any significant increases in tumors in this experiment.

Hansen et al. (1971) exposed Osborne-Mendel rats to 0, 5, 25, 125, 625, or 1,250 ppm 2,4-D in the diet for two years. There were 25 male and 25 female rats in each dosage group. No significant effect of dosage on survival was noted. Total numbers of rats with tumors in the control group was 15, and the tumors in the treated groups, by increasing dose, were 14, 18, 20, 23, and 22. Because the tumors were typical of those normally found in aging Osborne-Mendel rats and no target organ tumors were involved, the authors did not attribute these lesions to the feeding of 2,4-D. If one were to assume a relationship between dose and tumor incidence, statistical upper limits on the carcinogenic potency of 2,4-D can be calculated from the studies described above. These upper limits on the carcinogenic potency of 2,4-D are calculated in this analysis using a one-hit model of cancer. This model is the most conservative (i.e., predicts the highest risks) of any of the cancer models which have gained some acceptance. The one-hit model assumes no threshold or, in other words, it assumes that even a single molecule of a carcinogen might cause cancer. This model was used for a time by EPA to estimate cancer risks before being replaced by a less conservative multi-stage model of cancer (Crump 1983).

The one-hit model was fit separately to the male and female rat data on total animals with tumors from Hansen et al. (1971) using the computer program GLOBAL82 (Howe and Crump 1982). The data on females gave the largest 95 percent statistical upper limit on the carcinogenic potency of 2,4-D (i.e., largest 95 percent upper limit on the linear term in the one-hit model of cancer). This upper limit was 3.01×10^{-4} ppm^{-1} or 5.03×10^{-3} per (mg/kg/day) (Crump 1983). This carcinogenic potency factor is slightly higher than the factor calculated from the brain tumor data cited above.

The utility of the cancer potency factor is discussed at the end of this section. This factor is used to predict the probability of cancer in exposed populations.

The National Cancer Institute (NCI) has recently reported an epidemiologic study of Kansas farmworkers (Hoar et al. 1986). This study attempted to isolate the effect of specific herbicides including 2,4-D. This study reports that farmers who were exposed to phenoxyacetic acid herbicides (including 2,4-D) were about twice as likely as non-farmers to develop Non-Hodgkins Lymphoma (NHL), a rare form of cancer. Farmers who were exposed to phenoxyacetic acids over 20 days per year increased their odds of NHL sixfold. Farmers who were exposed over 20 days per year and did their own mixing and loading showed an eightfold increase.

Some findings of this study were inconsistent. For example, although farmers with increased exposure days per year had increased rates of NHL, there was no relation between increased acres sprayed per year and increased NHL rates. Likewise, an increase in the number of years of spraying was not associated with an increase in NHL rates. These inconsistencies may be reconciled by additional ongoing studies currently being conducted by the NCI.

The NCI study found no increase in NHL rates in farm family members who were not actively involved in herbicide application. Farmers who used personal protective measures were about 1.5 times as likely as non-farmers to develop NHL. Farmers who did not protect themselves were about 2.1 times as likely as non-farmers.

Because of the number of uncertainties in this NCI study, findings are not conclusive. Additional work is currently underway that could answer some of the questions raised in this study.

Several epidemiology studies conducted in Europe have indicated higher than expected cancer rates in workers exposed to 2,4-D in concert with other chemicals (see, for example, Barthel 1981, Ericsson et al. 1981, Lynge 1985). The results of these studies have been reviewed (USDA Forest Service 1987b). However, because the workers involved in these studies were exposed to a variety of chemicals, it is not possible to determine whether the excess cancer was due to the 2,4-D alone, the 2,4-D interacting with other chemicals, or some other chemical with no effect from the 2,4-D. Despite these uncertainties, the following conclusions reached by the review cited above regarding phenoxy herbicides seem appropriate.

> Suggestions of association with at least five types of cancer have been found in the epidemiology literature. Each of the five cancers has some statistically significant associations with exposure. While there is no conclusive demonstration of any individual association, the overall suggestion is that phenoxy herbicides in some way initiate or promote cancers, and that this occurs at a level of exposure experienced in various work settings.

The carcinogenic potential of various components of the mixture known as petroleum distillates has been reviewed (see USDA Forest Service 1987b). Petroleum distillates are contained in some 2,4-D formulations. The analysis concludes that the cancer potential, considering the small concentration of the compounds such as benzene and benzo(a)pyrene, is less than one-thousandth of that of the 2,4-D and would not add significantly to the potency of the 2,4-D mixture.

2.7.2.2 Picloram Cancer Studies

The data on the carcinogenic potential of picloram are ambiguous. The National Cancer Institute (1978) conducted a bioassay of picloram and interpreted the findings as "suggestive of ability of the compound to induce benign tumors in livers of female Osborne-Mendel rats." The benign lesion that suggested this effect was foci of cellular alteration in the liver. Whether such benign tumors could develop into true cancer is unclear. On the assumption that such a tumor could so develop, the one-hit model can be applied to data on this lesion in the manner described for 2,4-D. The 95 percent upper limit calculated in this fashion for the carcinogenic potency of picloram in 3.4×10^{-5} ppm or 5.68×10^{-4} per (mg/kg/day) (Crump 1983). This value is approximately one-tenth of the 2,4-D value.

2.7.2.3 Glyphosate Cancer Studies

The U.S. EPA is currently reviewing glyphosate carcinogenicity studies submitted by Monsanto (IBT replacement studies). Feeding studies (2-year) with both rats and mice have been conducted. Well-conducted rat studies showed no oncogenic activity in either sex. A mouse study is currently being reviewed by the U.S. EPA. In brief, this 2-year mouse oncogenicity (cancer) study was conducted with glyphosate feed levels of 1,000 parts per million (ppm) in food; 5,000 ppm in food; 30,000 ppm in food; and a control group. Each feed level was comprised of 50 animals of each sex.

The number of male mice with tumors (renal tubular adenomas) was 0 at the 1,000 ppm group, one in 5,000 ppm group, and three in the 30,000 ppm group. No females had tumors at any dose level. There is some controversy over whether there was one or 0 tumors in the male control (untreated) animals. The U.S. EPA has ordered Monsanto to recut and re-examine tissues from these animals to resolve the controversy. As noted in the 2,4-D studies, tumors in the control (untreated) mice are not unusual although tumors of the type found in this study have rarely been found in untreated mice.

In reviewing the oncogenicity studies of glyphosate, several conclusions can be drawn. First, these feeding studies reaffirm the relatively low toxicity of glyphosate. The highest dose levels of 30,000 ppm means that 3 percent of the mouse daily food intake was glyphosate.

Second, the weight of evidence as indicated by both mouse- and rat-feeding studies indicates, at most, weak oncogenic effect from glyphosate dose.

In summarizing the information on glyphosate oncogenicity, U.S. EPA (as cited in U.S. Department of Justice 1985) has concluded:

> Thus, in well-conducted oncogenicity studies on both sexes of two species, the incidence of only one tumor type in one sex of one species was found to have an increase related to treatment with glyphosate. This increase in tumors occurred only at very high exposure levels (much higher than usual in long-term studies of pesticides). Furthermore, the positive finding depends upon the presence of tumors in only four treated animals.
>
> The factors listed in the above paragraph indicate that the evidence for oncogenicity, though present, is extremely limited. According to the Agency's proposed carcinogen risk assessment guidelines (49 FR 46294), glyphosate would be classified in Category C which is used for agents with limited evidence of carcinogenicity in animals in the absence of human data. Category C is the lowest weight-of-evidence category among the categories with any positive evidence.
>
> In addition to the limited amount of quantitative evidence supporting a conclusion of oncogenicity, a quantitative risk estimate indicates that, to the extent that glyphosate is actually an oncogen, it is likely to have only a weak oncogenicity effect. This is primarily related to the extremely high doses at which effects were observed in the study as compared to likely human exposure. Therefore, based on the information currently available, the Agency does not expect any significant risk from the level of glyphosate to which humans are likely to be exposed.

This risk analysis assumes that glyphosate is a carcinogen. The 95 percent limit of the cancer potency calculated from the kidney tumor data is 3.4×10^{-5} per (mg/kg/day).

2.7.2.4 Amitrole Cancer Studies

The data on the carcinogenic potential of amitrole indicates carcinogenic effects in mammals exposed to amitrole. The U.S. EPA (1985b) has classified amitrole as a "probable human carcinogen." Amitrole cancer potency was estimated using data from three studies:

1. A 2-year rat-feeding study conducted by Hazleton Laboratories, Inc.

2. A study by Tsuda et al. (1976) in which rats were given 2,500 ppm in their drinking water.

3. A study by Food and Drug Research Laboratory (as cited in U.S. EPA 1985b) in which rats alternately were fed food with and without amitrole.

The cancer potency for amitrole estimated from the Hazleton Labs rat study data was 0.15 per (mg/kg/day) for all invasive thyroid lesions and 9.8×10^{-4} per (mg/kg/day) for papillary adenoma. The Food and Drug Research 1981 study (as cited in U.S. EPA 1985b) indicated a cancer potency for thyroid tumors of 0.61 (considering only the intermittently dosed groups). In this risk assessment, the greatest of these is used to estimate human cancer risk. The 95 percent upper confidence limit for the potency based on the Food and Drug Research data is 1.4 per (mg/kg/day).

2.7.2.5 Atrazine Cancer Studies

Recent test results submitted to the U.S. EPA by the manufacturer of atrazine indicate a carcinogenic effect in the animal feeding studies.

The results of this study can be summarized as follows (USDA, Forest Service 1986b):

> Technical grade atrazine was evaluated for chronic oral toxicity and oncogenicity in Sprague-Dawley rats fed dietary concentrations of 0 (untreated control), 10, 70, 500, and 1,000 ppm for two years. A number of chronic toxicity symptoms were observed: reduced body weight for animals in the 500 and 1,000 ppm test groups, reduce red cell parameters (such as red blood cell count and hemoglobin) for females in the 1,000 ppm test group, and decreased glucose levels in animals fed 1,000 ppm. These data indicate a systemic toxicity NOEL of 70 ppm.
>
> There was a significant increase in total mammary tumors in female rats fed 70, 500, and 1,000 ppm atrazine. These tumors were observed as tissue masses or lumps even before the animals were sacrificed for pathologic examination.
>
> Using the incidence of total mammary tumors (91 percent) in the 1,000 ppm group compared to the incidence of tumors (58 percent) in the untreated control, we estimate a cancer potency of 0.03 per (mg/kg/day) for atrazine using the single-hit cancer model described in the Gypsy Moth Final Environmental Impact Statement, as supplemented (USDA, Forest Service 1985).

Chronic tests of the herbicides hexazinone and dicamba (as reviewed in USDA, Forest Service 1984), indicate no carcinogenic potential for these compounds. No carcinogenic effects were found in 2-year feeding studies with mice or rats fed tebuthiuron at doses up to 1,600 ppm (USDA, Forest Service 1986a).

2.7.2.6 Cancer Prediction Models

The probability of the occurrence of cancer over a lifetime as a result of exposure to 2,4-D, picloram, amitrole, atrazine, or glyphosate can be assessed using the following equation:

$$Pc = q^* \times D \times De/L$$

where

Pc = the estimate of the probability of cancer as a result of the dose

q^* = the upper limit of the carcinogenic potency slope

5.03×10^{-3} per (mg/kg/day) for 2,4-D;

5.68×10^{-4} per (mg/kg/day) for picloram;

1.4 per (mg/kg/day) for amitrole;

3.4×10^{-5} per (mg/kg/day) for glyphosate; and

3.0×10^{-2} per (mg/kg/day) for atrazine

D = daily dose in mg/kg/day

De = number of days during which the daily dose occurs

L = days in a lifetime (25,550).

Using this equation, the incremental probability of cancer in a lifetime from each exposure pathway can be calculated for the model projects applying 2,4-D, picloram, glyphosate, amitrole, or atrazine. These probabilities are provided in Tables 2-97 through 2-127 for workers and for members of the general population. For example, the probability of a worker with a backpack sprayer developing cancer after spraying 2,4-D for 1 day (high dose, low protection) on mid-sized, projects is 5.2×10^{-8} (5.03×10^{-3} per (mg/kg/day) x 0.263 mg/kg/day x 1 day x lifetime/25,550 days). A cancer probability of 5.2×10^{-8} means that the worker has about five chances in one hundred million of developing cancer as a result of a one-day dose. The worker's probability of cancer as a result of 30 days' spraying, assuming he gets a high dose each day, is 1.6×10^{-6} or about 1 chance in a million. If the worker sprayed 30 days per year for 30 years, his additional cancer probability would be 4.7×10^{-5} or about 5 chances in 100,000.

Based on the finding of the NCI study of Kansas farmworkers cited above, the apparent odds of developing cancer as a result of exposure to herbicides is higher than predicted from animal test data. The rate of Non-Hodgkins Lymphoma in the general population in Kansas is about 1 in 10,000 (Holmes 1986). An odds ratio of 6 to 8 as noted to farmers exposed over 20 days per year would put their NHL rate in the vicinity of 6 to 8 per 10,000.

The calculation of the cancer probabilities for various members of the general population requires an estimate of the daily dose and the number of days over which the dose will occur. The maximum-exposed resident in the vicinity of a small project is assumed to receive a drift dose for 1 day, to consume drift-contaminated vegetables for 42 days and herbicide-contaminated beef for 140 days. The herbicide concentration on vegetation is assumed to remain constant for two weeks, to fall to one-half initial values for the next two weeks, and by another one-half for the next two weeks. It is possible that small amounts of the initial residues of persistent herbicides such as picloram could remain in the soil beyond the 42 days. The small incremental impact of these herbicide concentrations on cancer rates is adequately compensated for by the overestimation inherent in the use of a step function with a two-week interval to estimate concentrations that in reality would be falling continuously.

Assumptions similar to those above were made for residents and visitors in the vicinity of mid-sized and large projects except that 3 days' drift dose was assumed for residents near large projects. In addition to the routes of exposure for residents near these projects, residents near right-of-way and riparian projects were assumed to get a one-time dose from eating contaminated fish and drinking contaminated water in addition to eating contaminated beef and vegetables.

The cancer probabilities for the general population on Tables 2-98 through 2-127 (exclusive of worker tables) are provided for each exposure pathway and include consideration of dose duration (in days). For example, the cancer probabilities provided on Table 2-115 for a large project sprayed with 2,4-D are calculated as follows for an adult resident and visitor:

Drift:	probability = 1.1 x 10^{-10} = 0.00503 per (mg/kg/day) x 0.00018 mg/kg/day x 3 days x lifetime/25,550 days.
Oral dose, beef:	probability = 2.0 x 10^{-8} = 0.00503 per (mg/kg/day) x 0.00071 mg/kg/day x 140 days x lifetime/25,550 days.
Oral dose, vegetable:	probability = 2.3 x 10^{-8} = 0.00503 per (mg/kg/day) x ((0.0047 x 14 days) + 0.5(0.0047 x 14 days) + 0.25(0.0047 x 14 days) x lifetime/25550 days.
Visitor entry:	probability = 2.2 x 10^{-10} = 0.00503 per (mg/kg/day) x 0.0011 mg/kg/day x 1 day x lifetime/25,550 days.
Oral dose, wild food:	probability = 1.0 x 10^{-8} = 0.00503 per (mg/kg/day) x 0.051 mg/kg/day x 1 day x lifetime/25,550 days.

The cumulative impact on the maximum-exposed resident from doses from each of the exposure pathways is the sum of the probabilities from the individual pathways. For the maximum-exposed adult resident near a large project sprayed with 2,4-D (see Table 2-115), the cumulative cancer probability from all three exposure pathways is 4.3×10^{-8} ($1.1 \times 10^{-10} + 2.0 \times 10^{-8} + 2.3 \times 10^{-8}$) or about four chances in 100 million. If this resident were exposed to five projects in a lifetime and each time received the maximum doses (in itself a very, very low probability event), his probability of cancer would be 2.1×10^{-7} or about two chances in ten million.

Considering the herbicides that are widely used in the Northern Region and are presumed to be carcinogens (i.e., 2,4-D and picloram), the highest cancer probability occurs with an infant resident near a large project. The infant's cumulative cancer probability from use of 2,4-D is 5.9×10^{-8} or about six chances in 100 million. If the infant were exposed to worst-case doses from 10 projects in a lifetime, his additional cancer probability would be about six chances in ten million.

Exposure to amitrole and atrazine provides a higher probability of cancer than exposure to 2,4-D. Amitrole and atrazine probabilities are on the order of one in 100,000 to one in 1,000,000. These higher probabilities are primarily a result of a long-term (140 days) consumption of beef that is maximally contaminated with herbicides. As discussed in Section 2.6.1, the spraying of amitrole and atrazine for control of noxious weeds or poisonous plants is not likely in the Northern Region.

As a point of comparison and to further illustrate the reality of such small probabilities, Table 2-128 provides a list of events which result in a one-in-a-million chance of death. As shown on Table 2-128, the average American has about a one-in-a-million chance of being killed in fire for every 13 days of living in the U.S. His probability of a fire fatality for 1 year would be about 2.8×10^{-5} (1×10^{-6}/13 days x 365 days/year), or about three chances in 100,000. A worker in the transport and public utilities section of industry (e.g., a truck driver) has a one-in-a-million chance of death every day on the job. A person who smokes two cigarettes has increased his probability of cancer by one chance in a million.

Text continued on page 190.

Table 2-97. Cancer probabilities for backpack workers spraying small projects for 1 day (1.5 acres).

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	7.3×10^{-9}	1.46×10^{-8}	2.3×10^{-8}	4.6×10^{-8}
Picloram	8.9×10^{-11}	1.6×10^{-10}	2.5×10^{-10}	5.1×10^{-10}
Glyphosate	2.4×10^{-11}	4.9×10^{-11}	7.5×10^{-11}	1.5×10^{-10}
2,4-D/ Picloram	2.6×10^{-9} 2.2×10^{-11}	5.3×10^{-9} 4.5×10^{-11}	8.1×10^{-9} 6.7×10^{-11}	1.7×10^{-8} 1.3×10^{-10}
2,4-D/ Dicamba	2.6×10^{-9}	5.3×10^{-9}	8.1×10^{-9}	1.7×10^{-8}
Amitrole	2.2×10^{-7}	3.9×10^{-7}	6.0×10^{-7}	1.3×10^{-6}
Atrazine	2.11×10^{-8}	4.3×10^{-8}	6.7×10^{-8}	1.4×10^{-7}

Table 2-98. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with 2,4-D.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	9.1×10^{-12}	9.9×10^{-12}
Adolescent dermal dose	1.14×10^{-11}	1.24×10^{-11}
Infant dermal dose	2.13×10^{-11}	2.3×10^{-11}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.0×10^{-8}
Infant oral dose (beef)	2.3×10^{-8}	2.3×10^{-8}
Adult/adolescent oral dose (veg)	4.2×10^{-9}	4.6×10^{-9}
Infant oral dose (veg)	6.0×10^{-9}	6.5×10^{-9}
Visitor entry to spray site 1 day	2.2×10^{-11}	2.4×10^{-11}
Oral dose/sprayed wild food 1 day	1.0×10^{-8}	1.1×10^{-8}

Table 2-99. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with picloram.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	4.4×10^{-14}	6.7×10^{-14}
Adolescent dermal dose	6.7×10^{-14}	6.7×10^{-14}
Infant dermal dose	1.1×10^{-13}	1.3×10^{-13}
Adult/adolescent oral dose (beef)	2.2×10^{-9}	2.2×10^{-9}
Infant oral dose (beef)	2.6×10^{-9}	2.6×10^{-9}
Adult/adolescent oral dose (veg)	2.4×10^{-8}	2.6×10^{-8}
Infant oral dose (veg)	3.4×10^{-10}	3.6×10^{-10}
Visitor entry to spray site 1 day	2.5×10^{-13}	2.7×10^{-13}
Oral dose/sprayed wild food 1 day	5.8×10^{-10}	6.2×10^{-10}

Table 2-100. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with glyphosate.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	3.1×10^{-14}	3.3×10^{-14}
Adolescent dermal dose	3.9×10^{-14}	4.3×10^{-14}
Infant dermal dose	7.2×10^{-14}	7.9×10^{-14}
Adult/adolescent oral dose (beef)	1.3×10^{-10}	1.3×10^{-10}
Infant oral dose (beef)	1.5×10^{-10}	1.5×10^{-10}
Adult/adolescent oral dose (veg)	1.4×10^{-11}	1.6×10^{-11}
Infant oral dose (veg)	2.0×10^{-11}	2.2×10^{-11}
Visitor entry to spray site 1 day	7.05×10^{-14}	7.59×10^{-14}
Oral dose/sprayed wild food 1 day	3.5×10^{-11}	3.7×10^{-11}

Table 2-101. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with a 2,4-D/picloram mixture.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	$3.3 \times 10^{-12}/2.2 \times 10^{-14}$	$6.5 \times 10^{-12}/1.8 \times 10^{-14}$
Adolescent dermal dose	$4.3 \times 10^{-12}/2.2 \times 10^{-14}$	$4.7 \times 10^{-12}/2.2 \times 10^{-14}$
Infant dermal dose	$8.3 \times 10^{-12}/2.2 \times 10^{-14}$	$8.7 \times 10^{-12}/4.4 \times 10^{-14}$
Adult/adolescent oral dose (beef)	$2.0 \times 10^{-8}/2.2 \times 10^{-9}$	$2.0 \times 10^{-8}/2.2 \times 10^{-9}$
Infant oral dose (beef)	$2.3 \times 10^{-8}/2.6 \times 10^{-9}$	$2.3 \times 10^{-8}/2.6 \times 10^{-9}$
Adult/adolescent oral dose (veg)	$1.6 \times 10^{-9}/6.0 \times 10^{-11}$	$1.7 \times 10^{-9}/6.5 \times 10^{-11}$
Infant oral dose (veg)	$2.2 \times 10^{-9}/8.7 \times 10^{-11}$	$2.4 \times 10^{-9}/9.2 \times 10^{-11}$
Visitor entry to spray site 1 day	$7.9 \times 10^{-12}/6.6 \times 10^{-14}$	$8.3 \times 10^{-12}/6.6 \times 10^{-14}$
Oral dose/sprayed wild food 1 day	$3.7 \times 10^{-9}/1.4 \times 10^{-10}$	$4.1 \times 10^{-9}/1.6 \times 10^{-10}$

Table 2-102. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with a 2,4-D/dicamba mixture.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	3.3×10^{-12}	3.7×10^{-12}
Adolescent dermal dose	4.3×10^{-12}	4.7×10^{-12}
Infant dermal dose	8.3×10^{-12}	8.7×10^{-12}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.0×10^{-8}
Infant oral dose (beef)	2.3×10^{-8}	2.3×10^{-8}
Adult/adolescent oral dose (veg)	1.6×10^{-9}	1.7×10^{-9}
Infant oral dose (veg)	2.2×10^{-9}	2.4×10^{-9}
Visitor entry to spray site 1 day	7.9×10^{-12}	8.3×10^{-12}
Oral dose/sprayed wild food 1 day	3.7×10^{-9}	4.1×10^{-9}

Table 2-103. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with atrazine.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	5.4×10^{-11}	5.9×10^{-11}
Adolescent dermal dose	6.8×10^{-11}	7.4×10^{-11}
Infant dermal dose	1.3×10^{-10}	1.4×10^{-10}
Adult/adolescent oral dose (beef)	1.17×10^{-5}	1.17×10^{-5}
Infant oral dose (beef)	1.36×10^{-5}	1.36×10^{-5}
Adult/adolescent oral dose (veg)	1.3×10^{-8}	1.3×10^{-8}
Infant oral dose (veg)	1.78×10^{-8}	1.9×10^{-8}
Visitor entry to spray site 1 day	6.24×10^{-11}	6.7×10^{-11}
Oral dose/sprayed wild food 1 day	3.05×10^{-8}	3.29×10^{-8}

Table 2-104. Cancer probabilities for visitors and residents in the vicinity of a small project sprayed with amitrole.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	1.1×10^{-10}	1.6×10^{-10}
Adolescent dermal dose	1.6×10^{-10}	1.6×10^{-10}
Infant dermal dose	2.7×10^{-10}	3.3×10^{-10}
Adult/adolescent oral dose (beef)	5.4×10^{-6}	5.4×10^{-6}
Infant oral dose (beef)	6.4×10^{-6}	6.4×10^{-6}
Adult/adolescent oral dose (veg)	5.9×10^{-7}	6.4×10^{-7}
Infant oral dose (veg)	8.3×10^{-7}	9.0×10^{-7}
Visitor entry to spray site 1 day	6.03×10^{-10}	6.6×10^{-10}
Oral dose/sprayed wild food 1 day	1.4×10^{-6}	1.5×10^{-6}

Table 2-105. Cancer probabilities for one worker from spraying a mid-sized project (6 days).

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	5.0×10^{-8}	1.0×10^{-7}	1.5×10^{-7}	3.2×10^{-7}
Picloram	6.6×10^{-10}	1.1×10^{-9}	1.6×10^{-9}	3.4×10^{-9}
Glyphosate	1.6×10^{-10}	3.4×10^{-10}	5.2×10^{-10}	1.0×10^{-9}
2,4-D/ Picloram	1.8×10^{-8} 1.4×10^{-10}	3.6×10^{-8} 2.6×10^{-10}	5.4×10^{-8} 4.0×10^{-10}	1.1×10^{-7} 9.4×10^{-9}
2,4-D/ Dicamba	1.8×10^{-8}	3.6×10^{-8}	5.4×10^{-8}	1.1×10^{-7}
Amitrole	1.6×10^{-6}	2.6×10^{-6}	4.0×10^{-6}	8.6×10^{-6}
Atrazine	1.4×10^{-7}	3.0×10^{-7}	4.6×10^{-7}	9.2×10^{-7}

Table 2-106. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with 2,4-D.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	3.2×10^{-11}	3.2×10^{-11}
Adolescent dermal dose	3.9×10^{-11}	4.3×10^{-11}
Infant dermal dose	7.1×10^{-11}	7.9×10^{-11}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.0×10^{-8}
Infant oral dose (beef)	2.3×10^{-8}	2.3×10^{-8}
Adult/adolescent oral dose (veg)	7.4×10^{-9}	8.0×10^{-9}
Infant oral dose (veg)	1.0×10^{-8}	1.1×10^{-8}
Visitor entry to spray site 1 day	1.4×10^{-10}	1.5×10^{-10}
Oral dose/sprayed wild food 1 day	1.0×10^{-10}	1.1×10^{-10}

Table 2-107. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with picloram.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	8.9×10^{-14}	8.9×10^{-14}
Adolescent dermal dose	1.1×10^{-13}	1.1×10^{-13}
Infant dermal dose	2.0×10^{-13}	2.2×10^{-13}
Adult/adolescent oral dose (beef)	2.2×10^{-9}	2.2×10^{-9}
Infant oral dose (beef)	2.6×10^{-9}	2.6×10^{-9}
Adult/adolescent oral dose (veg)	4.2×10^{-10}	4.5×10^{-10}
Infant oral dose (veg)	5.8×10^{-10}	6.3×10^{-10}
Visitor entry to spray site 1 day	1.6×10^{-12}	1.8×10^{-12}
Oral dose/sprayed wild food 1 day	5.8×10^{-10}	6.2×10^{-10}

Table 2-108. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with glyphosate.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	5.3×10^{-14}	5.3×10^{-14}
Adolescent dermal dose	6.7×10^{-14}	6.7×10^{-14}
Infant dermal dose	1.2×10^{-13}	1.3×10^{-13}
Adult/adolescent oral dose (beef)	1.3×10^{-10}	1.3×10^{-10}
Infant oral dose (beef)	1.5×10^{-10}	1.5×10^{-10}
Adult/adolescent oral dose (veg)	2.5×10^{-11}	2.7×10^{-11}
Infant oral dose (veg)	3.5×10^{-11}	3.8×10^{-11}
Visitor entry to spray site 1 day	4.7×10^{-13}	5.1×10^{-13}
Oral dose/sprayed wild food 1 day	3.5×10^{-11}	3.7×10^{-11}

Table 2-109. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with a 2,4-D/picloram mixture.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	$5.9 \times 10^{-12}/2.2 \times 10^{-14}$	$5.9 \times 10^{-12}/2.2 \times 10^{-14}$
Adolescent dermal dose	$7.9 \times 10^{-12}/2.2 \times 10^{-14}$	$7.9 \times 10^{-12}/2.2 \times 10^{-14}$
Infant dermal dose	$1.4 \times 10^{-11}/4.4 \times 10^{-14}$	$1.4 \times 10^{-11}/4.4 \times 10^{-14}$
Adult/adolescent oral dose (beef)	$2.0 \times 10^{-8}/2.2 \times 10^{-9}$	$2.0 \times 10^{-8}/2.2 \times 10^{-9}$
Infant oral dose (beef)	$2.3 \times 10^{-8}/2.6 \times 10^{-9}$	$2.3 \times 10^{-8}/2.6 \times 10^{-9}$
Adult/adolescent oral dose (veg)	$2.8 \times 10^{-9}/1.0 \times 10^{-10}$	$3.0 \times 10^{-9}/1.2 \times 10^{-10}$
Infant oral dose (veg)	$3.9 \times 10^{-9}/1.4 \times 10^{-10}$	$4.2 \times 10^{-9}/1.7 \times 10^{-10}$
Visitor entry to spray site 1 day	$5.3 \times 10^{-11}/4.5 \times 10^{-13}$	$5.5 \times 10^{-11}/4.5 \times 10^{-13}$
Oral dose/sprayed wild food 1 day	$3.7 \times 10^{-9}/1.4 \times 10^{-10}$	$4.1 \times 10^{-9}/1.6 \times 10^{-10}$

Table 2-110. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with a 2,4-D/dicamba mixture.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	5.9×10^{-12}	5.9×10^{-12}
Adolescent dermal dose	7.9×10^{-12}	7.9×10^{-12}
Infant dermal dose	1.4×10^{-11}	1.4×10^{-11}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.0×10^{-8}
Infant oral dose (beef)	2.3×10^{-8}	2.3×10^{-8}
Adult/adolescent oral dose (veg)	2.8×10^{-9}	3.0×10^{-9}
Infant oral dose (veg)	3.9×10^{-9}	4.2×10^{-9}
Visitor entry to spray site 1 day	5.3×10^{-11}	5.5×10^{-11}
Oral dose/sprayed wild food 1 day	3.7×10^{-9}	4.1×10^{-9}

Table 2-111. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with atrazine.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	9.4×10^{-11}	9.4×10^{-11}
Adolescent dermal dose	1.2×10^{-11}	1.3×10^{-11}
Infant dermal dose	2.1×10^{-10}	2.3×10^{-10}
Adult/adolescent oral dose (beef)	1.2×10^{-5}	1.2×10^{-5}
Infant oral dose (beef)	1.4×10^{-5}	1.4×10^{-5}
Adult/adolescent oral dose (veg)	2.2×10^{-8}	2.4×10^{-8}
Infant oral dose (veg)	3.0×10^{-8}	3.3×10^{-8}
Visitor entry to spray site 1 day	4.1×10^{-10}	4.6×10^{-10}
Oral dose/sprayed wild food 1 day	3.0×10^{-8}	3.3×10^{-8}

Table 2-112. Cancer probabilities for visitors and residents in the vicinity of a mid-sized project sprayed with amitrole.

	Cancer probability assuming minor mixing error	Cancer probability assuming major mixing error
Adult dermal dose	2.2×10^{-10}	2.2×10^{-10}
Adolescent dermal dose	2.7×10^{-10}	3.0×10^{-10}
Infant dermal dose	4.9×10^{-10}	5.5×10^{-10}
Adult/adolescent oral dose (beef)	5.4×10^{-6}	5.4×10^{-6}
Infant oral dose (beef)	6.4×10^{-6}	6.4×10^{-6}
Adult/adolescent oral dose (veg)	1.0×10^{-6}	1.1×10^{-6}
Infant oral dose (veg)	1.4×10^{-6}	1.5×10^{-6}
Visitor entry to spray site 1 day	3.9×10^{-9}	4.4×10^{-9}
Oral dose/sprayed wild food 1 day	1.4×10^{-6}	1.5×10^{-6}

Table 2-113. Cancer probabilities for each backpack worker on a large project (10 days).

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	8.3×10^{-8}	1.7×10^{-7}	2.5×10^{-7}	5.2×10^{-7}
Picloram	1.1×10^{-9}	1.8×10^{-9}	2.67×10^{-9}	5.8×10^{-9}
Glyphosate	3.5×10^{-10}	5.6×10^{-10}	8.65×10^{-10}	1.7×10^{-9}
2,4-D/ Picloram	2.0×10^{-8} 2.2×10^{-10}	6.1×10^{-8} 4.5×10^{-10}	9.1×10^{-8} 6.7×10^{-10}	1.87×10^{-7} 1.6×10^{-9}
2,4-D/ Dicamba	2.0×10^{-8}	6.1×10^{-8}	9.1×10^{-8}	1.87×10^{-7}
Amitrole	2.7×10^{-6}	4.4×10^{-6}	6.6×10^{-6}	1.4×10^{-5}
Atrazine	2.4×10^{-7}	5.0×10^{-7}	7.6×10^{-7}	1.5×10^{-6}

Table 2-114. Cancer probabilities for a truck driver and a supervisor on a large project (10 days).

	Supervisor		Truck Driver	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	2.2×10^{-9}	9.5×10^{-9}	5.9×10^{-8}	2.2×10^{-7}
Picloram	6.7×10^{-12}	2.22×10^{-11}	1.6×10^{-10}	9.3×10^{-9}
Glyphosate	6.7×10^{-12}	3.2×10^{-11}	2.0×10^{-10}	7.5×10^{-10}
2,4-D/ Picloram	7.9×10^{-10} 6.7×10^{-12}	3.5×10^{-9} 2.2×10^{-11}	2.2×10^{-8} 1.6×10^{-10}	8.3×10^{-8} 6.7×10^{-10}
2,4-D/ Dicamba	7.9×10^{-10}	3.5×10^{-9}	2.2×10^{-8}	8.3×10^{-8}
Amitrole	5.5×10^{-8}	2.7×10^{-7}	1.6×10^{-6}	6.0×10^{-6}
Atrazine	5.9×10^{-9}	2.8×10^{-8}	1.8×10^{-7}	6.6×10^{-7}

Table 2-115. Cancer probabilities for visitors and residents in the vicinity of a large project sprayed with 2,4-D, picloram, or glyphosate.

	Cancer probability (2,4-D)	Cancer probability (picloram)	Cancer probability (glyphosate)
Adult dermal dose	1.1×10^{-10}	6.7×10^{-13}	3.6×10^{-13}
Adolescent dermal dose	1.4×10^{-10}	6.7×10^{-13}	4.8×10^{-13}
Infant dermal dose	2.5×10^{-10}	1.3×10^{-12}	8.4×10^{-13}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.2×10^{-9}	1.3×10^{-10}
Infant oral dose (beef)	2.3×10^{-8}	2.6×10^{-9}	1.5×10^{-10}
Adult/adolescent oral dose (veg)	2.3×10^{-8}	1.3×10^{-9}	7.8×10^{-11}
Infant oral dose (veg)	3.6×10^{-8}	2.0×10^{-9}	1.2×10^{-10}
Visitor entry to spray site 1 day	2.2×10^{-10}	2.2×10^{-12}	6.7×10^{-13}
Oral dose/sprayed wild food 1 day	1.0×10^{-8}	5.8×10^{-10}	3.5×10^{-11}

Table 2-116. Cancer probabilities for visitors and residents in the vicinity of a large project sprayed with a 2,4-D/picloram mixture.

	Probability from 2,4-D dose	Probability from picloram dose
Adult dermal dose	4.1×10^{-11}	1.3×10^{-13}
Adolescent dermal dose	5.3×10^{-11}	2.0×10^{-13}
Infant dermal dose	1.0×10^{-10}	3.3×10^{-13}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.2×10^{-9}
Infant oral dose (beef)	2.3×10^{-8}	2.6×10^{-9}
Adult/adolescent oral dose (veg)	8.7×10^{-9}	3.3×10^{-10}
Infant oral dose (veg)	1.4×10^{-8}	4.9×10^{-10}
Visitor entry to spray site 1 day	7.9×10^{-11}	6.7×10^{-13}
Oral dose/sprayed wild food 1 day	3.9×10^{-9}	1.3×10^{-10}

Table 2-117. Cancer probabilities for visitors and residents in the vicinity of a large project sprayed with a 2,4-D/dicamba mixture, amitrole, or atrazine.

	Probability from 2,4-D/dicamba mixture	Probability from amitrole dose	Probability from atrazine dose
Adult dermal dose	4.1×10^{-11}	1.6×10^{-9}	6.3×10^{-10}
Adolescent dermal dose	5.3×10^{-11}	1.6×10^{-9}	8.1×10^{-10}
Infant dermal dose	1.0×10^{-10}	3.3×10^{-9}	1.5×10^{-9}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	5.4×10^{-6}	1.2×10^{-5}
Infant oral dose (beef)	2.3×10^{-8}	6.4×10^{-6}	1.4×10^{-5}
Adult/adolescent oral dose (veg)	8.7×10^{-9}	3.2×10^{-6}	6.9×10^{-8}
Infant oral dose (veg)	1.4×10^{-8}	5.0×10^{-6}	1.1×10^{-7}
Visitor entry to spray site 1 day	7.9×10^{-11}	5.2×10^{-9}	5.9×10^{-10}
Oral dose/sprayed wild food 1 day	3.9×10^{-9}	1.4×10^{-6}	3.1×10^{-8}

Table 2-118. Cancer probabilities for a truck driver spraying a right-of-way and riparian project.

	Truck driver	
	Average	High
2,4-D	3.9×10^{-9}	1.6×10^{-8}
Picloram	4.5×10^{-11}	1.8×10^{-10}
Glyphosate	1.3×10^{-11}	5.3×10^{-11}
2,4-D/picloram	1.6×10^{-9} 1.1×10^{-11}	5.9×10^{-9} 5.9×10^{-11}
2,4-D/dicamba	1.6×10^{-9}	5.9×10^{-9}
Amitrole	1.1×10^{-7}	4.4×10^{-7}
Atrazine	1.2×10^{-8}	7.7×10^{-6}

Table 2-119. Cancer probabilities for a backpack worker on right-of-way and riparian projects (1 day).

	Recommended Protection		Low Protection	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	9.7×10^{-9}	1.2×10^{-8}	3.0×10^{-8}	6.1×10^{-8}
Picloram	1.1×10^{-10}	2.2×10^{-10}	3.3×10^{-10}	6.9×10^{-10}
Glyphosate	3.3×10^{-11}	6.5×10^{-11}	1.0×10^{-10}	2.1×10^{-10}
2,4-D/ Picloram	3.4×10^{-9} 2.2×10^{-11}	7.3×10^{-9} 4.5×10^{-11}	1.1×10^{-8} 8.9×10^{-11}	2.2×10^{-8} 1.6×10^{-10}
2,4-D/ Dicamba	3.4×10^{-9}	7.3×10^{-9}	1.1×10^{-8}	2.2×10^{-8}
Amitrole	2.7×10^{-7}	5.5×10^{-7}	8.2×10^{-7}	1.7×10^{-6}
Atrazine	2.9×10^{-8}	5.8×10^{-8}	8.9×10^{-8}	1.8×10^{-7}

Table 2-120. Cancer probabilities for visitors and residents in the vicinity of a right-of-way and riparian project sprayed with 2,4-D, picloram, or glyphosate.

	Probability from 2,4-D dose	Probability from picloram dose	Probability from glyphosate dose
Adult dermal dose	7.9×10^{-12}	4.2×10^{-14}	2.5×10^{-14}
Adolescent dermal dose	4.7×10^{-10}	5.3×10^{-12}	1.6×10^{-12}
Infant dermal dose	1.8×10^{-11}	1.0×10^{-13}	6.0×10^{-14}
Adult/adolescent oral dose (beef)	2.0×10^{-10}	2.2×10^{-11}	1.3×10^{-12}
Infant oral dose (beef)	2.3×10^{-10}	2.6×10^{-11}	1.5×10^{-12}
Adult/adolescent oral dose (veg)	2.4×10^{-9}	1.4×10^{-10}	8.1×10^{-12}
Infant oral dose (veg)	3.3×10^{-9}	1.9×10^{-10}	1.1×10^{-11}
Visitor entry or walk along ROW	1.1×10^{-10}	1.2×10^{-12}	3.7×10^{-13}
Adult oral dose (water)	1.1×10^{-9}	6.4×10^{-11}	3.8×10^{-12}
Adolescent oral dose (water)	1.5×10^{-9}	8.3×10^{-11}	5.0×10^{-12}
Infant oral dose (water)	1.6×10^{-9}	9.3×10^{-11}	5.5×10^{-12}
Adult/adolescent oral dose (fish)	1.9×10^{-11}	1.0×10^{-12}	6.3×10^{-14}
Infant oral dose (fish)	2.2×10^{-13}	1.2×10^{-14}	7.3×10^{-16}

Table 2-121. Cancer probabilities for visitors and residents in the vicinity of a right-of-way and riparian project sprayed with a 2,4-D/picloram mixture or atrazine.

	Probability from 2,4-D dose	Probability from picloram dose	Probability from atrazine dose
Adult dermal dose	2.8×10^{-12}	1.1×10^{-14}	4.5×10^{-11}
Adolescent dermal dose	1.7×10^{-10}	1.3×10^{-12}	1.4×10^{-9}
Infant dermal dose	6.7×10^{-12}	2.4×10^{-14}	1.1×10^{-10}
Adult/adolescent oral dose (beef)	2.0×10^{-10}	2.2×10^{-11}	1.2×10^{-7}
Infant oral dose (beef)	2.3×10^{-10}	2.6×10^{-11}	1.6×10^{-7}
Adult/adolescent oral dose (veg)	9.0×10^{-10}	3.4×10^{-11}	7.1×10^{-9}
Infant oral dose (veg)	1.3×10^{-9}	4.7×10^{-11}	9.9×10^{-9}
Visitor entry or walk along ROW	4.1×10^{-11}	5.5×10^{-12}	8.1×10^{-9}
Adult oral dose (water)	4.2×10^{-10}	1.6×10^{-11}	3.4×10^{-9}
Adolescent oral dose (water)	5.5×10^{-10}	2.1×10^{-11}	4.4×10^{-9}
Infant oral dose (water)	6.2×10^{-10}	2.3×10^{-11}	4.9×10^{-9}
Adult/adolescent oral dose (fish)	6.9×10^{-12}	2.7×10^{-13}	1.1×10^{-8}
Infant oral dose (fish)	8.1×10^{-12}	3.1×10^{-15}	3.2×10^{-10}

Table 2-122. Cancer probabilities for visitors and residents in the vicinity of right-of-way and riparian project sprayed with a 2,4-D/dicamba mixture or amitrole.

	Probability from 2,4-D/dicamba mixture	Probability from amitrole dose
Adult dermal dose	2.8×10^{-12}	1.0×10^{-10}
Adolescent dermal dose	1.7×10^{-10}	1.3×10^{-8}
Infant dermal dose	6.7×10^{-12}	2.5×10^{-10}
Adult/adolescent oral dose (beef)	2.0×10^{-10}	5.4×10^{-8}
Infant oral dose (beef)	2.3×10^{-10}	6.4×10^{-8}
Adult/adolescent oral dose (veg)	9.0×10^{-10}	3.3×10^{-7}
Infant oral dose (veg)	1.3×10^{-9}	4.6×10^{-7}
Visitor entry or walk along ROW	4.1×10^{-11}	7.5×10^{-8}
Adult oral dose (water)	4.2×10^{-10}	1.6×10^{-7}
Adolescent oral dose (water)	5.5×10^{-10}	2.1×10^{-7}
Infant oral dose (water)	6.2×10^{-10}	2.3×10^{-7}
Adult/adolescent oral dose (fish)	6.9×10^{-12}	2.6×10^{-9}
Infant oral dose (fish)	8.1×10^{-12}	3.0×10^{-9}

Table 2-123. Cancer probabilities for pilots and mixer/loaders on aerial spray projects.

	Pilot		Mixer/Loader	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	3.3×10^{-9}	1.4×10^{-8}	3.7×10^{-9}	1.0×10^{-8}
2,4-D/Dicamba	1.2×10^{-9}	4.9×10^{-9}	1.4×10^{-9}	3.7×10^{-9}
Picloram	4.4×10^{-11}	1.6×10^{-10}	4.4×10^{-11}	1.1×10^{-10}

Table 2-124. Cancer probabilities for supervisors and observers on aerial spray projects.

	Supervisor		Observer	
	Average Dose	High Dose	Average Dose	High Dose
2,4-D	5.9×10^{-10}	2.0×10^{-9}	9.9×10^{-11}	2.8×10^{-10}
2,4-D/Dicamba	1.8×10^{-10}	7.9×10^{-10}	3.9×10^{-11}	9.8×10^{-11}
Picloram	6.7×10^{-12}	2.7×10^{-11}	1.1×10^{-12}	3.1×10^{-12}

Table 2-125. Cancer probabilities for visitors and residents in the vicinity of a project sprayed aerially with 2,4-D.

	Worst-Case Condition	Routine Condition
Adult dermal dose	3.9×10^{-11}	3.9×10^{-14}
Adolescent dermal dose	5.1×10^{-11}	5.1×10^{-14}
Infant dermal dose	9.5×10^{-11}	9.5×10^{-14}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.0×10^{-8}
Infant oral dose (beef)	2.3×10^{-8}	2.3×10^{-8}
Adult/adolescent oral dose (veg)	3.2×10^{-8}	3.2×10^{-11}
Infant oral dose (veg)	4.9×10^{-8}	4.9×10^{-11}
Adult oral dose (water)	6.7×10^{-9}	1.3×10^{-10}
Adolescent oral dose (water)	5.9×10^{-9}	1.2×10^{-10}
Infant oral dose (water)	9.9×10^{-9}	2.0×10^{-10}
Visitor entry	2.0×10^{-9}	5.9×10^{-10}
Oral dose/sprayed wild food	1.0×10^{-8}	1.0×10^{-8}

Table 2-126. Cancer probabilities for visitors and residents in the vicinity of a project sprayed aerially with picloram.

	Worst-Case Condition	Routine Condition
Adult dermal dose	2.2×10^{-13}	2.2×10^{-16}
Adolescent dermal dose	2.9×10^{-13}	2.2×10^{-16}
Infant dermal dose	5.3×10^{-10}	4.4×10^{-16}
Adult/adolescent oral dose (beef)	2.2×10^{-9}	2.2×10^{-9}
Infant oral dose (beef)	2.6×10^{-9}	2.6×10^{-9}
Adult/adolescent oral dose (veg)	1.8×10^{-9}	1.8×10^{-12}
Infant oral dose (veg)	2.8×10^{-9}	2.8×10^{-12}
Adult oral dose (water)	3.8×10^{-10}	7.5×10^{-12}
Adolescent oral dose (water)	3.3×10^{-10}	6.7×10^{-12}
Infant oral dose (water)	5.6×10^{-10}	1.1×10^{-11}
Visitor entry	2.7×10^{-11}	6.7×10^{-12}
Oral dose/sprayed wild food	5.8×10^{-10}	5.8×10^{-10}

Table 2-127. Cancer probabilities for visitors and residents in the vicinity of a project sprayed aerially with 2,4-D/dicamba.

	Worst-Case Condition	Routine Condition
Adult dermal dose	1.5×10^{-11}	1.6×10^{-14}
Adolescent dermal dose	1.9×10^{-11}	2.0×10^{-14}
Infant dermal dose	3.5×10^{-11}	3.9×10^{-14}
Adult/adolescent oral dose (beef)	2.0×10^{-8}	2.0×10^{-8}
Infant oral dose (beef)	2.3×10^{-8}	2.3×10^{-8}
Adult/adolescent oral dose (veg)	1.1×10^{-8}	1.1×10^{-11}
Infant oral dose (veg)	1.6×10^{-8}	1.6×10^{-11}
Adult oral dose (water)	2.4×10^{-9}	4.7×10^{-11}
Adolescent oral dose (water)	2.0×10^{-9}	3.9×10^{-11}
Infant oral dose (water)	3.3×10^{-9}	6.7×10^{-11}
Visitor entry	7.9×10^{-10}	1.8×10^{-10}
Oral dose/sprayed wild food	3.9×10^{-9}	3.9×10^{-9}

Table 2-128. Lifetime risk of death or cancer resulting from everyday activities.

Activity	Time to accumulate a one-in-a-million risk of death	Average annual risk per capita
	Living in the United States	
Motor vehicle accident	1.5 days	2×10^{-4}
Falls	6 days	6×10^{-5}
Drowning	10 days	4×10^{-5}
Fires	13 days	3×10^{-5}
Firearms	36 days	1×10^{-5}
Electrocution	2 months	5×10^{-6}
Tornados	20 months	6×10^{-7}
Floods	20 months	6×10^{-7}
Lightning	2 years	5×10^{-7}
Animal bite or sting	4 years	2×10^{-7}
	Occupational Risks	
General		
manufacturing	4.5 days	8×10^{-5}
trade	7 days	5×10^{-3}
service & government	3.5 days	1×10^{-4}
transport & public utilities	1 day	4×10^{-4}
agriculture	15 hours	6×10^{-4}
construction	14 hours	6×10^{-4}
mining and quarrying	9 hours	1×10^{-3}
Specific		
coal mining (accidents)	14 hours	6×10^{-4}
police duty	1.5 days	2×10^{-4}
railroad employment	1.5 days	2×10^{-4}
fire fighting	11 hours	8×10^{-4}

Other One-In-A-Million Risks

Source of risk	Type and amount of exposure: examples
Cosmic rays	One transcontinental round trip by air; living 1.5 months in Colorado compared to New York; camping at 15,000 feet over 6 days compared to sea level.
Other	20 days of sea level natural background radiation; 2.5 months in masonry rather than wood building; 1/7 of a chest x-ray using modern equipment.
Eating & drinking	40 diet sodas (saccharin) 6 pounds of peanut butter (aflatoxin) 180 pints of milk (aflatoxin) 200 gallons of drinking water from Miami or New Orleans 90 pounds of broiled steak (cancer risk only)
Smoking	2 cigarettes

[1] From Crouch and Wilson (1982)

2.7.3 Risk of Heritable Mutations

To date epidemiologic or other human studies have not linked exposure to any of these herbicides with heritable mutations (U.S. EPA 1984a). Nor has EPA accepted any quantitative systems for weighing the evidence on the mutagenicity of a compound or for conducting risk assessments.

For several of the herbicides reviewed above, mutagenicity tests conducted are insufficient to conclude whether the chemical can cause heritable mutations. For these herbicides, a worst-case assumption is made that these herbicides have the potential to cause mutations in humans. In these cases, the results of carcinogenicity tests or cancer risk assessments can be used to estimate the risk of heritable mutations. The rationale for this assumption is summarized by the USDA (1985) as follows: "Since mutagenicity and carcinogenicity both follow similar mechanistic steps (at least those that involve genetic toxicity), the increased risk of cancer can be used to approximate the quantitative risk of heritable mutations. The basis for this assumption is that both mutagenicity and at least primary carcinogens react with DNA to form a mutation or DNA lesion affecting a particular gene or set of genes. The genetic lesions then require specific metabolic processes to occur, or the cells must divide to insert the lesion into the genetic code of the cell. We believe the cancer risk provides a worst-case approximation to heritable mutations because cancer involves many types of cells whereas heritable mutations involve only germinal (reproductive) cells."

2.8 SYNERGISM/CUMULATIVE EFFECTS

This section examines the interaction of these herbicides with other chemicals in the environment and the cumulative effect of these programs on the herbicide present in the environment from other sources. Synergism, which concerns many people, is a special type of interaction where the combined effect of a specific herbicide with one or more chemicals in the environment (such as pollutants) would be greater than the sum of the individual effects of the herbicide and chemical(s) (in other words 2 + 2 are greater than 4).

Chemical interactions may also result in antagonistic effects in which two or more chemicals cause opposite effects on the same physiologic function or decrease the intrinsic activity of one of the components. Some cases of chemical interactions lead to a decrease in toxicologic activity, which is one of the common principles of antidotal treatment. Examples include the use of chelating agents to bond with metal ions and the use of ammonia as an antidote to the ingestion of formaldehyde.

Since there are many chemicals in use, the interaction of herbicides with other chemicals in the environment is possible. However, because of the complex number of possible interactions, the result is not readily predictable.

One way to measure interactive effects is to conduct epidemiological studies on exposed and control populations. However, the interactive effects described are often measurably small and the sensitivity of epidemiology tests might not be sufficient to detect such effects, particularly at the dose levels occurring with most treatment programs and with the small number of people involved.

A classic study of the synergistic effects of pollutants examined the interactive effects of asbestos exposure and smoking. Selikoff et al. (1968) found that inhalation of cigarette smoke and asbestos resulted in an eightfold increase in lung cancer over nonsmokers exposed to only asbestos. Studies such as these, however, have limitations because high doses are required to discover effects and their relevance to low-level exposures is uncertain.

Tests for synergistic effects can sometimes be accomplished using short-term animal or cellular tests at relatively high dosage levels. For example, Statham and Lech (1975a, 1975b) have reported the synergistic effects of the pesticide carbaryl on the acute toxicity of 2,4-D in trout, as well as the pesticides dieldrin, rotenone, and pentachlorophenol. The acute toxicity of these chemicals was increased by factors of threefold to about eightfold for additions of 1 mg/liter of carbaryl. This amount of carbaryl is much higher than would be present in water under any circumstance except worst-case accident scenarios.

Some mixtures of picloram and 2,4-D are reported to cause dermal sensitization in a small percentage of the human population, and precautionary statements to this effect are contained on the label for Tordon 101 (trademark for a mixture of the triisopropanolamine salts of picloram and 2,4-D). Since these compounds are not reported to cause dermal sensitization reactions singly, there may be an interactive effect from this mixture. Tank mixes (i.e., mixed in the field) of the potassium salt of picloram and the amine salts of 2,4-D have not been reported to cause dermal sensitization in the field but such mixtures have not been specifically studied in the laboratory.

In summary then, what can be said concerning the issue of synergistic and cumulative effects from herbicides relative to the Forest Service herbicide plant control programs?

First, the additive impact of Forest spraying on top of general effects of the private application of herbicides will be very small. For example, a worker or farmer who sprays herbicides on non-Forest Service projects and who is also a resident in the vicinity of Forest Service projects might expect, under worst-case conditions, an increase in herbicide dose of about 1 percent over his worker dose (see discussion in Section 2.6). Typically, the increase would not be measurable.

Second, the total doses to members of the general public from all sources of herbicides are unlikely to be higher than those estimated in these analyses. The dose to maximum-exposed residents assumed that the greatest portion of their diet came from spray-impacted foodstuffs. Any substitution of food from other sources (i.e., food markets) would lessen the dose. The herbicides involved in these analyses have not been found widely in market foodstuffs. For example, a market-basket analysis by the Natural Resources Defense Council (NRDC) of a variety of fruits and vegetables found no 2,4-D in any food sample (NRDC 1984).

Although the NRDC (1984) found other pesticides in some foodstuffs, the interactive effects would be suspected to be small for maximum-exposed residents. Since the dose or concentration of any chemical dictates both the probability and rate of any chemical reaction (and all biological responses in an organism are the result of chemical reactions), the dose of a specific herbicide in the environment or in the individual is an important factor in considering synergistic effects. Ames (1983) pointed out that there are many naturally occurring chemicals in the food people eat which are teratogenic, mutagenic, and carcinogenic and which are consumed at doses 10,000 times higher than man-made herbicides. Therefore, the low, short-lived doses to maximum-exposed residents that result from spraying of these herbicides to control target plants are very small compared to many other chemicals in the environment. For these small comparative doses, a synergistic effect is not realistically expected (Crouch et al. 1983). The EPA apparently came to the same conclusion, because they issued a Notice (PR Notice 82-1) on January 12, 1982 (U.S. EPA 1982a) rescinding the requirement for submission of tank mix compatibility data. The Notice stated that EPA had examined considerable data and found no evidence of potentiation involving herbicides.

As discussed throughout these analyses, the highest doses are expected of some types of workers, particularly those involved in hand application of herbicides. In some cases, workers will apply mixtures of herbicides, particularly 2,4-D/dicamba and 2,4-D/picloram. If one assumed synergistic reactions on the order of those observed in the case of asbestos exposure and smoking, then eight to tenfold increases in herbicide toxicity might be expected. The most significant impacts might involve workers spraying 2,4-D/dicamba mixtures. Again, the major concern would be the potential fetotoxic effects on pregnant female applicators. A ten-fold increase in the fetotoxic and reproductive toxic effects of dicamba would bring high worker doses close to levels that affected reproductive functions in laboratory animals and would increase the risks discussed in Section 2.6.2.

3.0 MAJOR ACCIDENT SCENARIOS

3.1 BACKGROUND

Several types of major accidents, their probability of occurrence, and the possible exposure of human populations are analyzed in this section.

Accidents such as large spills at the mixing/loading site or into potable water sources could result in worst possible direct and indirect human exposures. Direct exposure results primarily from spray or liquid deposition on the skin or from immersion into the liquid. Indirect exposure results from the consumption of contaminated water supplies or food.

The impact of a spill or dump depends on many variables such as the spill source (truck, aircraft, or backpack), size of load, distance to water, stream size, and density of human population. In order to bracket the impact of a spill of herbicide into a water supply reservoir, the possible impact on two different reservoirs was investigated. The first assumed a spill into a large reservoir which is a major source of drinking water for a town of 35,000. A second spill was assumed in a small reservoir which was the sole source of drinking water for a community of 500 residents. In developing the accident scenarios, extreme assumptions were used on all critical parameters.

The effects of a second type of worst-case accident involving an aircraft spill were also analyzed. In this scenario, a helicopter was assumed to jettison its load over workers at an application site. Direct exposure to the spilled herbicide would result in dermal absorption of the herbicide deposited on those at the spill site.

Analyses were also made of various other types of vehicle and personal accidents. None of these other accident types would involve as great a potential for human exposure and dosage as the worst-case scenarios outlined above.

3.2 TRUCK SPILLS

3.2.1 Probability of Occurrence

Several sources were reviewed for information on frequency of accidents involving herbicides. Personal injury/illness reports and vehicle accident reports for the Northern Region for the years 1977-1986 were reviewed. These records indicate that there were no accidents involving Forest Service vehicles transporting any type of pesticide including herbicides for plant control programs during that time period.

Calculation of the probability of a vehicle accident in which a major spill of herbicide is released in water or on land, is based on U.S. Department of Transportation (DOT) accident statistics for single-unit trucks, the type commonly used in plant control projects, as opposed to large tractor-trailer trucks.

According to the DOT Highway Statistics Division, single-unit trucks traveled 353,978 million miles in 1981. National Accident Sampling System (NASS) statistics estimate that single-unit trucks were involved in 162,000 accidents that year, or 1 accident for every 2,185,049 miles traveled (353,978 million miles/162,000 accidents). The mean probability of a single-unit truck accident can be calculated:

P_a = 1 per 2,185,049 miles per accident = 0.000000458 or 4.6×10^{-7} accidents per mile

where

P_a = the mean probability of a single-unit truck accident per mile.

The frequency of accidents differs according to road type. The mean probability of a single-unit truck accident can subsequently be adjusted to take road type into account. The following tabulation gives total miles traveled, number of accidents, and accident frequency (miles traveled per accident) for single-unit truck accidents for road type based upon 1981 data.

The probability of an accident occurring per mile is the inverse of the accident frequency as follows:

	Single-unit truck		Accident frequency (miles traveled per accident)	Probability of accident/mile
Road type	Total miles (million)	Number of accidents		
Urban interstate	23,059	13,449	1,714,551	5.8×10^{-7}
Rural interstate	28,758	958	30,018,789	3.3×10^{-8}
Other urban roads	146,195	92,430	1,581,683	6.3×10^{-7}
Other rural roads	155,966	55,163	2,827,366	3.5×10^{-7}

It is estimated that single-unit Forest Service trucks used on target plant control projects traveled road types in the following proportions:

Other urban roads - 10 percent.
Other rural roads - 90 percent.

By applying the accident probabilities for road type just generated to the proportions traveled during plant control projects, an adjusted probability of occurrence for single-unit truck accidents can be calculated as follows:

$$P_a = (0.10 \times 6.3 \times 10^{-7}) + (0.90 \times 3.5 \times 10^{-7}) = 0.000000378 \text{ or } 3.8 \times 10^{-7} \text{ per mile}$$

where

P_a = probability of an accident involving a single-unit truck occurring per mile traveled.

Not all accidents will result in the release of herbicide. In estimating the potential for herbicide release, accident severity must be taken into account. As noted earlier, accident estimates provided thus far include all accidents reported to authorities regardless of severity. In adjusting for probability of herbicide release, it is assumed that only those accidents severe enough to require towing vehicles from the scene of an accident result in the release of herbicide.

The only data base available on the severity of accidents aggregates single and tandem axle trucks together, although size of the load is categorized. For these vehicles, 68 percent were involved in collisions with other vehicles, 21 percent with fixed objects, and 10 percent were noncollision accidents. Towing was required in 20 percent of the multi-vehicle collisions, 60 percent of the collisions with fixed objects, and 100 percent of the turnovers and ruptures.

The probability of a truck accident resulting in herbicide release can be calculated for each accident type:

$$P = P_a \times A_t \times P_t$$

where

P_a = probability of an accident occurring per mile traveled (3.8×10^{-9})

A_t = proportion of accidents by accident type (0.68, 0.21, and 0.10)

P_t = proportion of accidents by accident type that require towing (0.2, 0.6, and 1.0)

For example, for accidents that involve collision with another vehicle, this computes as:

$$p = (3.8 \times 10^{-7}) \times (0.68) \times (0.2) = 5.2 \times 10^{-8}$$

The probability of herbicide release for all truck accident types is summarized below:

Accident type	Probability of release
Collision with vehicle	$p = 5.2 \times 10^{-8}$
Collision with fixed object	$p = 4.8 \times 10^{-8}$
Noncollision accident	$p = 3.8 \times 10^{-8}$
TOTAL	$p = 1.4 \times 10^{-7}$

The probability of a truck accident releasing herbicide for all accident types is the sum of the individual probabilities or, $P = 1.4 \times 10^{-7}$ per mile traveled.

Assuming that a vehicle carrying herbicide travels an average of 30 loaded miles during the course of a project for each of the approximately 1,220 projects expected in a year in the Northern Region, the annual probability that a traffic accident would occur in which herbicide is spilled would be 1.4×10^{-7}

accidents/mile x 30 miles/project x 1,200 projects/year or 5.0×10^{-3} accidents/year. On the average then, about five accidents every 1,000 years might be expected to result in a spill of herbicides.

Generally most trucks carrying herbicide would be carrying small quantities (4 to 8 pounds active ingredient) to supply backpack spray projects. From a health effects perspective, the trucks carrying the largest quantities are the greatest concern. Trucks carrying large quantities of diluted herbicide spray mixture (up to 200 gallons or 757 liters) are typically involved in spraying travelway projects. Assuming these trucks drive an average of 40 miles per project and for 30 of these miles they are loaded with herbicide (or conversely 10 miles empty or returning to reload), and assuming 100 projects per year, the probability of an accident resulting in a spill of herbicide from these larger trucks would be 4.2×10^{-4} (30 miles/project x 100 projects/year x 1.4×10^{-7} accidents/mile). This is equivalent to a about one accident involving a herbicide spill every 2,400 years.

3.2.2 Worst-Case Truck Spill

As demonstrated in the previous section, the probability of a major spill of herbicide is relatively small. Nonetheless, the small probability cannot be denied. It is, of course, impossible to predict the exact nature, effect, or frequency of such an occurrence. In order to place a boundary on the impact of accidents involving herbicides, an analysis of the worst-case type of accident was performed. If the risk to human health can be shown for such an accident, then it is reasonable to expect that the health effects from less catastrophic incidents should be less.

A worst-case truck spill is hypothesized to involve the rupture of a tank carrying 200 gallons (757 liters) of pesticide mixture containing 22 pounds (10 kilograms) of herbicide active ingredient (a.i.).

It is assumed that the worst place to spill a large quantity of herbicide would be into a domestic water supply reservoir. A worst-case reservoir is assumed to be one in which a spill will result in the highest dose over time to the greatest number of people. The highest concentration for the longest period of time would occur in those reservoirs with small volumes and a long hydraulic residence time (i.e., low flow through the reservoir). These characteristics, however, tend to be mutually exclusive since low volume reservoirs will necessarily have higher throughput (short residence time).

Two extreme model cases were selected for further analysis. The first analysis was based on a spill of herbicide into a large reservoir serving a large number of people. The second scenario assumed a spill into a smaller reservoir serving fewer people. In the first case, larger numbers of people would be exposed to smaller concentrations because the large reservoir would provide substantial dilution. In the second scenario, the impact of higher dosage to smaller populations was analyzed.

In the first spill scenario, it is assumed that 200 gallons (757 liters) of herbicide mixture containing 22 pounds (10 kilograms) of herbicide, a.i. is spilled directly into a reservoir which provides 35 to 50 percent of the drinking water for a city with a population of 35,000 (e.g. Butte, Montana). As is typical of larger towns and cities in the Northern Region area, the affected town is assumed to be supplied simultaneously by several sources of drinking water. Although such a city could shut down one water source if problems develop, this analysis assumes that the city continues to pump from the contaminated reservoir.

The accident scenario assumes that herbicide is spilled directly into the reservoir and that the herbicide mixes and is available in a very short period for uptake at the water supply intake. It is also assumed that no biological degradation, hydrolysis, or chemical oxidation of the compound occurs and that the concentration is reduced only through dilution. The change in concentration over time from dilution by clean inflow water can be simulated by the differential equation:

$$1.\quad \frac{dH}{dt} = \frac{-QH}{V}$$

where:

H = mass of herbicide in the reservoir (milligrams)
Q = low summer flow to reservoir (liters/day)
V = volume of the reservoir
t = time (days)

The solution for the equation (1) is:

$$2.\quad H = A \exp\left(\frac{-Qt}{V}\right)$$

where A = H at initial conditions (t = 0)

The reservoir is assumed to be at a summer low volume of 200 million gallons (760 million liters) and receiving a summer low flow of 1.9 million gallons (7.2 million liters) per day.

Using equation 2 it can be shown that from an initial concentration of 0.013 mg/liter, the concentration would fall to 0.005 mg/liter in 100 days if dilution were the only method of removal. Again, this concentration is an overestimation because photolytic oxidation, hydrolysis, biological degradation, and adsorption to sediments would also contribute to the reduction of the herbicide concentration.

Assuming that the water is diluted 50 percent with water from other sources and that the average 70 kg person consumes 2 liters of water per day, the dose of herbicide on the initial day would be 0.00019 mg/kg. This dose is below the acceptable daily intake (ADI) for all herbicides analyzed here. The dose is more than 50 times below the ADI for 2,4-D which has the lowest ADI of the herbicides being considered.

The impact of a worst-case accidental spill of the herbicides picloram, 2,4-D, glyphosate, amitrole, and atrazine on cancer rates can also be calculated. To calculate dosage, it was assumed that the concentration would fall with dilution as outlined above for 270 days, at which time high spring time runoff would completely flush the reservoir of the remaining low concentration, thus effectively ending exposure. Assuming only dilution, the concentration in the reservoir would fall to 0.001 mg/L by day 270.

In order to calculate the total dosage to consumers of the water over the 270-day exposure period, each day is treated as a separate exposure event. The mass of herbicide contained in the reservoir on each of the exposure days can be summed by integrating equation 2 between t = 0 and t = 270.

$$TH = A \int_{t = 0}^{t = 270} \exp\left(\frac{-Qt}{V}\right) = A \Big|_{t = 0}^{t = 270} \frac{\exp\left(\frac{-Qt}{V}\right)}{\frac{-Q}{V}}$$

Using this integral, the total mass of herbicide (TH) summed over the time frame would be 9.7×10^8 mg. The average daily concentration can be calculated as 0.0047 mg/L (9.7×10^8 mg/270 days/7.6×10^8 L). The average dose to the average individual (70 kg individual) over the 270 days (assuming 50 percent dilution of contaminated water) would be 6.7×10^{-5} mg/kg (0.0047 mg/L x 2 L x 0.5 x 1/70 kg).

As discussed in Section 2.7.9, the probability of cancer occurring to a person as a result of this exposure can be calculated using the equation:

$$Pc = q^* \times D \times De/L$$

where

Pc = worst-case estimate of the probability of cancer as a result of the dose

q* = the upper limit of the carcinogenic potency slope

5.03×10^{-3} per (mg/kg/day) for 2,4-D;
5.68×10^{-4} per (mg/kg/day) for picloram;
3.4×10^{-5} per (mg/kg/day) for glyphosate;
1.4 per (mg/kg/day) for amitrole;
3.0×10^{-2} per (mg/kg/day) for atrazine.

D = average daily dose in mg/kg/day

De = number of days during which the daily dose occurs
L = days in a lifetime (25,550).

Using this equation, the incremental probability of cancer in a lifetime from drinking contaminated water can be calculated.

For glyphosate, this probability of cancer for an individual is 2.4×10^{-11}, or about three chances in 100 billion (.000034 per (mg/kg/day) x .000067 mg/kg x 270/25,550). For the entire population of 35,000, the probability of an additional case of cancer is about 8.4×10^{-7}, or about one chance in a million ($35{,}000 \times 2.4 \times 10^{-11}$).

For picloram, this probability of cancer for an individual, given the worst-case accident scenario, is 4.0×10^{-10} in a 70-year lifetime or four chances in 10 billion. For the entire population of 35,000, the probability of an additional case of cancer is 1.4×10^{-5} ($35{,}000 \times 4.0 \times 10^{-10}$) or about one chance in 100,000.

For 2,4-D, the probability of cancer for an individual, given the worst-case accident scenario, is 3.6×10^{-9} in a 70-year lifetime (or about four chances in one billion). For the entire population of 35,000, the probability of an additional case of cancer is 1.2×10^{-4} or about one chance in 10,000.

For amitrole, the probability of cancer for an individual, given the worst-case accident scenario, is 9.9×10^{-7} or about one chance in a million. For the entire population of 35,000, the probability of an additional case of cancer is 3.5×10^{-2} or about one chance in 30.

For atrazine, the probability of cancer for an individual, given the worst-case scenario, is 2.1×10^{-8} in a 70-year lifetime (or about 2 chances in 100 million). For the entire population of 35,000, the probability of an additional case of cancer is 7.4×10^{-4} or about 7 chances in 10,000.

The impact of a major spill into a smaller reservoir serving a community of 500 residents can also be analyzed. This reservoir is assumed to have a capacity of 10 million liters (2.3 million gallons) and a low daily summer inflow of 3.8 million liters (1.0 million gallons).

Using the same initial spill conditions and other assumptions presented above, it is possible to calculate the change in concentration over time. From an initial concentration of 0.5 milligrams per liter, the concentration would fall to 0.07 mg/L in 10 days and 0.011 mg/L in 20 days (assuming only dilution). Based on a 2-liter per day consumption of water, the dose on the first day to a 70 kg person would be 0.014 mg/kg. This initial dose is below the ADI for atrazine, glyphosate, picloram, and tebuthiuron. Within one day, assuming only dilution, the dose would be below the ADI for all herbicides (except amitrole, which does not have an ADI). In addition to dilution, many mechanisms such as photo decomposition, bacterial decomposition, and attachment to colloids would reduce the herbicide concentration.

In calculating the effects of such a spill, it is evident that the greatest impacts would occur from concentrations experienced in the first 40 days and that the incremental impacts after that would be insignificant in comparison.

The concentrations in the reservoir on day 40 would be 0.00025 mg/L (from 0.5 mg/L on day 1). The total daily doses after day 40 could be expected to be less than 0.1 percent of the total daily doses up to that day. Thus a 40-day exposure period is used in determining cancer rates from possible exposure to glyphosate, picloram, 2,4-D, amitrole, and atrazine.

As in the previous example, the total mass of herbicide over the 40 days was calculated from the integral:

$$TH = A \int_{t=0}^{t=40} \exp\left(\frac{-Qt}{V}\right) = A \left. \frac{\exp\left(\frac{-Qt}{V}\right)}{\frac{-Q}{V}} \right|_{t=0}^{t=40}$$

The total mass of herbicide (TH) integrated over the 40 days would be 5.2×10^7 mg. The average daily concentration would be 0.065 mg/L (5.2×10^7 mg/40 days/20×10^6 L). The average daily dose to a 70 kg person drinking 2.0 liters of water per day would be 1.9×10^{-3} mg/kg (0.065 mg/L x 2 L x 1/70 kg).

As with the previous example, the probability of cancer to an individual and to the entire population can be calculated. For picloram, the probability of cancer for an individual given the worst-case accident scenario is 1.7×10^{-9} or about two chances in one billion. For the entire exposed population of 500 people, the total probability of an additional case of cancer over the lifetime of all residents following the spill is 8.0×10^{-7} ($500 \times 1.7 \times 10^{-9}$) or about one chance in a million.

For glyphosate, the probability of cancer for an individual, given the worst-case accident scenario, is 7.1×10^{-11} or about seven chances in 100 billion. For the entire exposed population of 500 people, the probability for an additional case of cancer following the accidental spill is 3.6×10^{-8} ($7.1 \times 10^{-11} \times 500$) or about four chances in 100 million.

For 2,4-D, the probability of cancer for an individual, given the worst-case accident scenario, is 1.5×10^{-8} or about 1.5 chances in 100 million. For the entire exposed population of 500 people, the probability of an additional case of cancer following the spill is 7.5×10^{-6}.

For amitrole, the probability of cancer for an individual is 2.2×10^{-6} or about two chances in one million. For the entire exposed population of 500 residents, the probability of an additional case of cancer is 1.1×10^{-3} or about one chance per thousand.

For atrazine, the probability of cancer for an individual is 8.9×10^{-8} or about one chance in 10 million. For the entire exposed population of 500 residents, the probability of an additional case of cancer over the 70-year period following the spill is 4.5×10^{-5} or about five chances per 100,000.

In comparing the two hypothetical truck spills, it can be seen that slightly higher probabilities of cancer occur to individuals in the smaller community. However, a larger number of people would be exposed in the larger community. Thus, there is a higher chance that someone in the larger community will develop cancer. By analogy, if every person in Moab, Utah, had 10 chances in a lottery and everyone in New York City had only one chance in the lottery, your chances of winning (as an individual) are greater in Moab. However, it is more likely that the winner will come from New York.

3.2.3 Probability of a Worst-Case Truck Spill

As might be expected, the probability of a worst-case truck spill is very small, the intersection of several rare events. As discussed in Section 3.2.1, trucks carrying large amounts of herbicide could be expected to be involved in an accident resulting in the spill of herbicides only once every 2,400 years.

The probability that a serious accident could impact a water supply system can be calculated by estimating the proportion of driving in the vicinity of water supply reservoirs. It was assumed that the 128 above-ground community water systems in the Northern Region would each have approximately 25 miles of streamside road upstream of the reservoir. This is an overestimate since many of these systems draw water from protected, unroaded, or minimally roaded watersheds. Using the 25-mile estimate, a total of 3,200 miles of road would be present in the upstream vicinity of water supply systems. There are 37,000 miles of Forest Service roads and approximately 9,000 miles of State and county roads on the National Forests in the Northern Region.

Assuming, conservatively, that all reservoirs and the roads above reservoirs are on National Forest System land, seven percent of National Forest roads would thus be in the vicinity of reservoirs. Assuming that roads near reservoirs are driven with equal frequency as all other roads (a conservative assumption in the Northern Region since many reservoirs are isolated and not accessible by heavily traveled thoroughfares), the probability of a serious truck accident on a road in the vicinity of a water supply system is thus reduced to one accident every 34,000 years (one major accident/2,400 years/.07). In addition, if the spill occurred on land, harm to persons served by the water supply would be further reduced.

3.3 WORST-CASE AIRCRAFT SPILL

This section presents data on possible impacts of spills from aircraft involved in aerial application of herbicides. It should be noted that the Forest Service has never aerially applied herbicides to control noxious weeds in the Northern

Region and has no plans to do so in the future. Indeed, in the past less than 50 acres a year have been aerially sprayed to control noxious weeds on all National Forests in western Regions.

No accident data is available from the limited aerial spray programs to control target plants. In order to analyze the risks from aerial application programs, data from a Pacific Southwest Region (California) program involving extensive aerial application of herbicides for commercial timber site preparation will be used to estimate the probability and potential impacts of accidents. Since the site preparation program often involves more severe terrain and operating conditions than other plant control programs, accident data from the California program would be expected to overestimate the probability and impact of aircraft accidents from range projects.

Several additional facts will be helpful to the reader in interpreting the possible impacts of aerial application programs. The analysis presumes the use of helicopter application because of the small size of most control projects. An advantage to this application method is that the helicopter can be landed at the application site with all herbicide mixing and loading occurring on-site. Aerial transport is thus minimized, as is the probability of spills over nontarget areas.

Reference will be made in the following section to the possibility of a helicopter jettisoning a load of herbicide. The Federal Aviation Administration requires that low-powered helicopters rigged for aerial applications have the capacity to jettison a full load under emergency conditions. The jettison time for a typically equipped fully loaded helicopter is approximately three seconds.

3.3.1 Probability of Occurrence

A review of herbicide aircraft incident records from the Pacific Southwest Region site preparation and release programs indicates that six total incidents involving aircraft occurred from 1976 through 1983. One incident involved a crash with spillage of 150 gallons of diluted spray mixture. A second incident involved a helicopter boom hitting a tree, breaking the boom with no loss of herbicide. Two incidents involved the jettison of material (250 gallons and 1 gallon), although there was no subsequent crash, and two incidents involved the overspray of streams.

Total acreage treated during those years was 148,000. Assuming 35 percent of these acres were aerially sprayed (or 51,800 acres) and 12 acres were treated per aerial load, then 4,320 individual aerial trips were required.

From this data, the probability of an aircraft incident per flight is six per 4,320 or 0.00014. The probability of occurrence of a major spill per flight is 2/4,320 or 0.00046. This is one per 2,160 flights.

An upper limit for the accident frequency (λ_1) can be calculated by a method described by Thedeen (1979) if the accidents are assumed to occur randomly in time. If N(a) is the number of occurrences for up to "a" events (trips, miles driven, etc.), the upper confidence level with a 1-α confidence limit can be calculated as follows:

$$\lambda_1 = \frac{X^2_\alpha}{2a}$$

Where X^2_α is the standard chi square distribution found in statistical tables and summarized below for n equals 2(N(a)+1).

	$2\lambda_1$	a	
n	= 0.500 (50)	= 0.05 (95)	= 0.01 (99)
2	1.39	5.99	9.2
4	3.36	9.49	13.2
6	5.35	12.6	16.8
8	7.34	15.5	20.1
10	9.34	18.3	23.2
12	11.3	21.0	26.2
14	13.3	23.7	29.1

For six incidents (N(a) = 6) in 4,320 aircraft loads, the value of X^2_α 95 confidence limit is 23.7 and the accident frequency, λ_1 , is calculated:

$$\lambda_1 = 23.7/(2 \times 4{,}320)$$
$$= .0027$$

for two major spills (N(a) = 2) in 4,320 aircraft loads, the value of X^2_α for the 95 confidence limit is 12.6 and the accident frequency, λ_1 , is calculated:

$$\lambda_1 = 12.6/(2 \times 4{,}320)$$
$$= .0015$$

Thus, the upper limit on accident rates indicates that there are 1.5 chances out of a thousand that any helicopter spray trip would involve a major spill.

3.3.2 Worst-Case Aircraft Spill

For the type of helicopter under consideration, the maximum aircraft load at high elevations common to the Northern Region is approximately 120 gallons (454 liters) of mixed herbicide. Each aerial load could cover from 10 to 20 acres (4-8 hectares). Assuming intended coverage of 11 acres (4.5 hectares) at 1 lb/ac (1.1 kg/ha), and mixing and formulation errors of 10 percent and 4 percent, respectively, the helicopter would be carrying 12.5 pounds (4.5 kilograms) of herbicide a.i. (5.7 ha x 1.1 kg/ha x 1.04 x 1.1).

At the time of the postulated jettison, the helicopter is presumed to be traveling at 30 mi/hr (48 km/hr), and to drop its load over four workers involved in mixing/loading and supervision at the spray site. Based on a jettison time of three seconds and a speed of 13.3 m/sec (48 km/hr), the spill is presumed to cover an area 40 meters long and 6 meters wide or 240 square meters.

Approximately 23.75 grams a.i. of herbicide would be deposited per square meter of spill area. Each worker is assumed to have 2 ft^2 (0.18 m^2) of uncovered skin exposed directly to the spill. The worker is also assumed to have 6 ft^2 (0.56 m^2) of clothing exposed to the spill. Twenty-five percent of the herbicide absorbed in clothing is assumed to contact the skin. As discussed in Section 2.4, this analysis assumes dermal absorption rates of 1 percent for amitrole and picloram; 10 percent for glyphosate, dicamba, and 2,4-D; and 20 percent for hexazinone and atrazine. Tebuthiuron is not included in the spill scenario because it is only applied as pellets.

The worst-case dose of glyphosate or 2,4-D would be 10.9 mg/kg (23,750 mg/m^2 x .18 m^2 x .1 x 1/70 kg) + (23,750 mg/m^2 x .56 m^2 x .25 x .1 x 1/70 kg). The doses of all other herbicides under these worst-case conditions are provided in Table 3-1. Cancer probabilities to a worker resulting from a one-time exposure to these doses are also provided in Table 3-1 for picloram, 2,4-D, amitrole, and atrazine. The methodology outlined in Section 2.6 is used for these calculations.

The doses provided on Table 3-1 are above the NOEL values for all the herbicides except picloram. Generally, such doses would be of short duration. Because the effects from a dose level decrease with a decrease in exposure period, the effects from a one-time dose would likely be slight to nonexistent. However, the medical literature does report rare instances of significant health effects such as peripheral neuropathy from large exposures to herbicides such as 2,4-D (see discussion in Section 2.6.1).

3.3.3 Probability of Worst-case Aerial Exposure

As demonstrated in Section 3.3.1, the upper limit of the probability of a spill is about 1.5×10^{-3} per flight or about 1.5 spills every 1,000 trips. The probability that a major spill would directly expose workers cannot be calculated except to say that it is much smaller than the probability of an accident. The greatest possibility of exposure to people would occur in the vicinity of the loading zone during take-off. All personnel typically evacuate this area during helicopter take-off and the helicopter flies from the area quickly.

3.4 OTHER ACCIDENT EXPOSURE SCENARIOS

Several other accidental exposure scenarios were examined as discussed below. In all cases, the exposure would result in human health impacts that are no more severe than those discussed under the worst-case accident scenarios.

In the event of a major truck accident and subsequent herbicide spills, there exists the possibility that the driver and cleanup workers could be directly exposed to herbicide. However, the exposure would be no greater than that detailed in the worst-case aerial spill directly over workers.

Worker exposure could result in the event of the spill of backpack application carrying 3 gallons of mixed herbicide. Direct exposure to a worker carrying the backpack would likewise be no greater than that presumed in the worst-case aerial spill.

The spill of 120 gallons of mixed herbicide from an aircraft into a domestic water supply reservoir would result in lower concentrations than that detailed in the worst-case truck spill since the aircraft would be carrying a smaller quantity of herbicide active ingredient.

Table 3-1. Worst-case doses and cancer probabilities from dermal exposure from an aerial spill.[1/]

	Dose (mg/kg)	Cancer probability
Picloram	1.3	2.9×10^{-8}
2,4-D	26.1	5.1×10^{-6}
Glyphosate	13.1	1.7×10^{-8}
Dicamba	13.1	---
Amitrole	1.3	7.2×10^{-5}
Atrazine	26.1	3.1×10^{-5}
Hexazinone	26.1	---

[1/] Assumes 1.2 kg/ha application rate and jettison of the herbicide directly over the bystander.

4.0 GLOSSARY

ACCEPTABLE DAILY INTAKE (ADI): The maximum dose of a substance that is anticipated to be without lifetime risk to humans when taken daily.

ACID EQUIVALENT (A.E.): The amount of active ingredient expressed in terms of the parent acid.

ACTIVE INGREDIENT (A.I.): The pesticide compound or toxicant which produces the desired effect of the formulation. Pesticide formulations are typically 1 to 50 percent active ingredient; the remainder being carriers, solvents, emulsifiers, etc.

CARCINOGEN: Any cancer-producing substance.

CARRIER: Material added to an active ingredient to facilitate its preparation, storage, shipment, or use.

CHRONIC TOXICITY: The poisoning effects of a series of small doses applied over a long period.

CONCENTRATION: The amount of active ingredient or acid equivalent in a quantity of diluent, expressed as lb/gal, ml/liter, etc.

DERMAL EXPOSURE: The contact of a chemical with skin.

DOSE: A given quantity of test material that is taken into the body; quantity of material to be administered.

EMULSIFIABLE CONCENTRATE: A liquid formulation obtained by dissolving the technical active ingredient in a liquid solvent and adding one or more emulsifiers, so that the formulated pesticide can be further diluted with water or oil for spray application.

EXPOSURE: Application of test material to the external surfaces of a test organism; takes into consideration route, duration, and frequency.

FETOTOXIC: Capable of producing adverse effects in a developing fetus.

FORMULATION: (1) A pesticide preparation supplied by a manufacturer for practical use.

(2) A manufacturing process by which technical active ingredients are prepared for practical use by mixing with liquid or dry diluents, grinding, or by the addition of emulsifiers, stabilizers, and other adjuvants.

HERBICIDE: A chemical used to control, suppress, or kill plants, or to severely interrupt their normal growth processes.

LC: Lethal concentration.

LC_{50}: The median lethal concentration; the concentration of toxicant necessary to kill 50 percent of the organisms being tested. It is usually expressed in parts per million (ppm).

LD_{50}: The median lethal dose; the size of a single dose of a chemical necessary to kill 50 percent of the organisms in a specific test situation. It is usually expressed in the weight of the chemical per unit of body weight (mg/kg). It may be fed (oral LD_{50}) or applied to the skin (dermal LD_{50}).

MUTAGENIC: Capable of inducing a mutation. An agent (change in hereditary material) that tends to increase the occurrence or extent of mutation.

NOEL: In a series of dose levels tested, it is the highest level at which no effect is observed (no-observed effect level).

NONTARGET VEGETATION: Vegetation which is not expected or not planned to be affected by the treatment.

ONCOGENIC (TUMORIGENIC): Capable of producing or inducing tumors in animals. The tumors may be either malignant (cancerous) or benign (noncancerous).

PESTICIDE: As defined by U.S. EPA, any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest and any substance or mixture of substances intended for use as a plant regulator, defoliant, or desiccant.

PHEROMONE: Any substance secreted by an animal which influences the behavior of other individuals of the same species.

RATE: The amount of active ingredient or acid equivalent applied per unit area or other treatment unit.

RESIDUE: That quantity of herbicide, its degradation products, and/or its metabolites remaining on or in the soil, plant parts, animal tissues, whole organisms, and surfaces.

RISK: The probability that a substance will produce harm under specified conditions.

SAFETY: The reciprocal of risk, i.e., the probability that harm will not occur under specified conditions.

SPOT TREATMENT: A herbicide applied over a small continuous restricted area of a whole unit; i.e., treatment of spots or patches or brush within a larger field.

SUBCHRONIC TOXICITY: Effects of regularly repeated doses or exposures over periods ranging from a few days to several months.

TERATOGEN: Any substance capable of producing structural abnormalities of prenatal origin, present at birth or manifested shortly thereafter (the ability to produce birth defects).

TOXICITY: The capacity or property of a substance to cause any adverse effects. It is based on scientifically verifiable data from animal or human exposure tests.

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